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1888. SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
1890. TIMOTHY HOLMES

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(Limited to Twelve.)

Elected

1887 FLOWER, WILLIAM HENRY, C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 FOSTER, MICHAEL, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the French Institute; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; The Camp, Sunningdale.

1868 HUXLEY, THOMAS HENRY, LL.D., D.C.L., F.R.S., late Professor of Natural History in the Royal School of Mines; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Hayes, Kent.

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, East Sheen, Mortlake.

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1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; President of the Royal Society; Lensfield Cottage, Cambridge.

1887 TURNER, SIR WILLIAM, LL.D., D.C.L., F.R.S., Professor of Anatomy in the University of Edinburgh.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Honorary Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Hind Head House, Shotter Mill, near Petersfield.

FOREIGN HONORARY FELLOWS.

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1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.

1883 BIGELOW, HENRY J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1887 BILLINGS, JOHN S., M.D., D.C.L. Oxon., Surgeon U.S.Army; Librarian, Surgeon-General's Office, Washington.

1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; 20, Alger Strasse, Vienna.

1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1883 DUBOIS REYMOND, EMIL, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

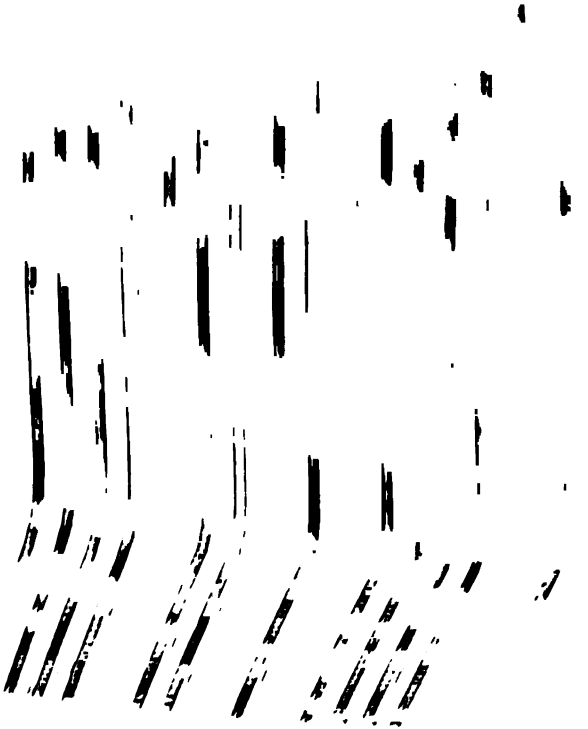
1887 ESMARCH, FRIEDRICH, M.D., Professor of Surgery in the University of Kiel.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 VON HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.



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SEPTEMBER, 1890.

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Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D.

1877 †ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

1851 *ACLAND, SIR HENRY WENTWORTH, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad street, Oxford.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton; 7, Brook street, Hanover square.

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., LL.D.Glasgow, F.R.S., Commissioner in Lunacy; Consulting Physician to the Leeds General Infirmary; 3, Melbury Road, Kensington. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Surgeon to the General Hospital, 5, East Circus Street, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., Assistant Physician to the London Hospital and to the National Hospital for the Paralysed and Epileptic; 41, Wimpole street, Cavendish square.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; 105, Gloucester place, Portman square.

1862 ANDREW, JAMES, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

1880 *APPLETON, HENRY, M.D., Staines.

Elected

1888 ARKLE, CHARLES JOSEPH, M.B.

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Honorary Physician to H. M. the Queen; Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1887 BALL, JAMES BARRY, M.D., 54, Wimpole street, Cavendish square.

1885 BALLANCE, CHARLES ALFRED, M.S., Senior Assistant Surgeon, West London Hospital; Assistant Surgeon, Hospital for Sick Children, Great Ormond street; Assistant Surgeon for Skin Diseases, St. Thomas's Hospital; 56, Harley street, Cavendish square. *Trans.* 1.

1848 †BALLARD, EDWARD, M.D., F.R.S., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Sci. Com.* 1889. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, SIR JOHN THOMAS, M.D., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

Elected

1886 BANKS, WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1879 BARKER, ARTHUR EDWARD JAMES, Surgeon to University College Hospital, and Assistant Professor of Clinical Surgery and Teacher of Practical Surgery at University College, London; 87, Harley street, Cavendish square. *Trans.* 6.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D., B.S., Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond street, and to the London Fever Hospital; 10, Wimpole street, Cavendish square. *Trans.* 2.

1881 *BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., 15, Harley street, Cavendish square. C. 1877-8. V.P. 1889–90. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital, to the Westminster Hospital, and to the West London Hospital; 17, Welbeck street, Cavendish square.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 11.

Elected

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. *Referee*, 1886-8. C. 1885. *Trans.* 2.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886-7. *Referee*, 1882-5. *Lib. Com.* 1881-5.

1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.

1871 BELLAMY, EDWARD, Senior Surgeon to, and Lecturer on Surgery at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; Examiner in Surgery in the Victoria University, Manchester; 17, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1879-81. *Trans.* 1.

1847 BENNET, JAMES HENRY, M.D., Mentone, Alpes Maritimes, France.

Elected

1880 BENNETT, ALEX. HUGHES, M.D., Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.

1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17 George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 1, Chesterfield street, Mayfair. *Trans.* 4.

1889 BENTLEY, ARTHUR J. M., M.D., 9, Somers place, Hyde Park.

1890 BERRY, DAVID ANDERSON, M.B., C.M., 117, Goldhawk Road.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 60, Welbeck street, Cavendish square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

1856 †BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

Elected

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D.

1881 BISS, CECIL YATES, M.D., Asisstant Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1846 †BOSTOCK, JOHN ASHTON, C.B., *Treasurer;* Hon. Surgeon to H.M. the Queen; Deputy Surgeon-General; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888-90. *Sci. Com.* 1867.

1890 BOSTOCK, R. ASHTON, 73, Onslow gardens, Brompton.

1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City]; Arcachon, France.

1882 BOWLBY, ANTHONY ALFRED, Surgical Registrar to St. Bartholomew's Hospital; 75, Warrington crescent, Maida hill. *Trans.* 3.

1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone. *Trans.* 1.

1841 †BOWMAN, SIR WILLIAM, BART., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

Elected

1886 BOXALL, ROBERT, M.D., Physician to the General Lying-in Hospital; 6, Chandos street, Cavendish square.

1884 BOYD, STANLEY, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, Charing Cross Hospital; 134, Harley street, Cavendish square.

1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

1890 BRADFORD, JOHN ROSE, M.B., D.Sc., 52, Upper Berkeley street, Portman square.

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 BRADSHAW, JAMES DIXON, M.B., 30, George Street, Hanover square.

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., Harrow-on-the-Hill, Middlesex.

1890 BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4. *Trans.* 5.

1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

Elected

1888 BROWNE, HENRY LANGLEY, Moor House, West Bromwich.

1878 BROWNE, SIR JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 7, Norland place, Holland Park.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Sci. Com.* 1889. *Referee*, 1886-8. *Lib. Com.* 1888. *Trans.* 1.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7.

1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Corresponding Member "Société de Chirurgie, Paris;" 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 11. *Pro.* 1.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 27, Woburn square.

1864 BUCKLE, FLEETWOOD, M.D.

1889 BULL, WILLIAM CHARLES, M.B., 35, Clarges street, Piccadilly.

1831 BULLER, AUDLEY CECIL, M.D.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook street, Grosvenor square.

Elected

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Trans.* 3.

1871 BUTT, WILLIAM F., 1, Southwick crescent, Hyde Park.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Women, Soho Square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee.* 1887-8.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1890 CAGNEY, JAMES, M.D., 11, Welbeck street, Cavendish square. *Trans.* 1.

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.

1888 CARLESS, ALBERT, M.B., B.S., Assistant Surgeon to King's College Hospital; 15, Stratford place, Oxford street.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1888 CARTER, WILLIAM JEFFREYS BECHER.

1845 †CARTWRIGHT, SAMUEL, Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON.

1888 CAUTLEY, EDMUND, M.B., B.C., 15, Upper Brook street.

Elected

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 60, Great Cumberland place. *Lib. Com.* 1885-8. *Trans.* 7.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Albert Mansions, 106, Victoria street, Westminster.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple row, Birmingham. *Trans.* 3.

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Senior Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890. *Sci. Com.* 1889. *Referee*, 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., Surgeon to King's College Hospital, and Demonstrator of Surgery in King's College, London; 59, Welbeck street, Cavendish square. *Lib. Com.* 1886-8.

1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.

Elected

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., C.I.E., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. *Referee*, 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., *Trustee*, *Vice-President*, Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875. V.P. 1888.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Miller Hospital, and Senior Assistant Surgeon to the Central London Ophthalmic Hospital; 21, Lee terrace, Blackheath.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1888 CLARKE, ROBERT HENRY, M.B., Clarence Lodge, Redhill, Surrey.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, C.M.G., Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

Elected

1879 †CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester Square.

1868 COCKLE, JOHN, M.D., F.L.S., Consulting Physician to the Royal Free Hospital; 8, Suffolk street, Pall Mall. *Trans.* 2.

1885 COLLINS, WILLIAM MAUNSELL, M.D., 10, Cadogan place.

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Surgeon to the Lock Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1868 CORNISH, WILLIAM ROBERT, C.I.E., late Surgeon-General, Madras Army; Hon. Physician to H.M. the Queen; 8, Cresswell gardens, The Boltons.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Ormean Terrace, Belfast.

1889 COSENS, CHARLES HENRY, St. Bartholomew's Hospital.

1860 †COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square.

1862 †COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., Hon. Surgeon to H.M. the Queen; late Director-General, Army Medical Department; 5, St. John's Park, Blackheath. C. 1887.

Elected

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

1862 CROMPTON, SAMUEL, M.D., Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1890 CROWLE, THOMAS HENRY RICKARD, 3, Campden Hill road.

1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital, and to the Great Northern Hospital; 17, Queen Anne street, Cavendish square.

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884-8.

1886 DAKIN, WILLIAM RADFORD, M.D., 57, Welbeck street, Cavendish square.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 18, Savile row. *Trans.* 3.

1884 DALLAWAY, DENNIS, 5, Duchess street, Portland place.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.D., Physician to the Radcliffe Infirmary, Oxford.

Elected

1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

1882 *DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1889 DEAN, HENRY PERCY, M.B., B.S., 60, Gower street.

1889 DELÉPINE, SHERIDAN, B.S., M.B., 6, Chapel place, Cavendish square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Trans.* 3.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867-79, 1889. *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

Elected

1889 DODD, HENRY WORK, 47, Kensington Park gardens.

1845 DODD, JOHN.

1888 DONELAN, JAMES, M.B., M.C., 2, Upper Wimpole street, Cavendish square.

1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Trans.* 1.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, M.B., B.S., The Small House, Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

1885 DRUMMOND, DAVID, M.D., 7, Saville Place, Newcastle-on-Tyne.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.

1865 †DUCKWORTH, SIR DYCE, M.D., Physician in Ordinary to H.R.H. the Prince of Wales; Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-8. *Trans.* 2.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

Elected

1871 DUKE, BENJAMIN, Windmill House, Clapham Common.

1871 *DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

1889 *DUNCAN, JOHN, M.D., St. Petersburg, Russia.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon and Pathologist at the West London Hospital; 2, Henrietta street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V. P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., 82, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 34, Leinster square.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.

Elected

1883 EDWARDES, EDWARD JOSHUA, M.D., 16, Acacia road, St. John's Wood.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 93, Wimpole street, Cavendish square.

1824 EDWARDS, GEORGE.

1887 ELLIOTT, JOHN.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 ELLISTON, WILLIAM ALFRED, M.D., Manor House, Ipswich.

1842 †ERICHSEN, JOHN ERIC, LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-7. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Assistant Surgeon to the London Hospital; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. *Sci. Com.* 1889. *Trans.* 1.

1875 *FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde park; Finzean, Aboyne Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

Elected

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, India Office; Physician to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.

1887 FEENY, MICHAEL HENRY, Les Avants, Montreux, Switzerland.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-8. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; 34, Wimpole street, Cavendish square.

1879 FINLAY, DAVID WHITE, M.D., Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley street, Portman square. *Trans.* 2.

1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 DE FONMARTIN, HENRY, M.D., 1, Anchor Gate terrace, Portsea, Hants.

Elected

1865 FOSTER, SIR BALTHAZAR WALTER, M.D., M.P., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.

1883 FOWLER, JAMES KINGSTON, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1887 FOX, RICHARD HINGSTON, M.D., 23, Finsbury square.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1871 FRANK, PHILIP, M.D., Cannes, France.

1884 *FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin. *Trans.* 1.

1843 FRASER, PATRICK, M.D., C. 1866.

1889 FREEMAN, HENRY WILLIAM, 24, The Circus, Bath.

1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, May Fair.

1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

Elected

1874 †GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. *Referee*, 1882-8. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.

1885 GAMGEE, ARTHUR, M.D., F.R.S.

1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886-8. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

1854 †GARROD, SIR ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889. *Trans.* 3.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

1889 *GASKELL, WALTER HOLBROOK, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; Petersfield House, Parkside, Cambridge.

1819 GAULTER, HENRY.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

1866 GEE, SAMUEL JONES, M.D., *Librarian*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-90. *Sci. Com.* 1879. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

Elected

1885 GELL, HENRY WILLINGHAM, M.B., 43, Albion street, Hyde Park.

1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884-8. *Trans.* 1.

1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Referee*, 1886-8. *Trans.* 5.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 12, Queen Anne street, Cavendish square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1889 GOODSALL, DAVID HENRY, 17, Devonshire place, Upper Wimpole street.

Elected

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Referee* 1888. *Lib. Com.* 1884-6. *Trans.* 7.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1889 GREENE, GEORGE EDWARD JOSEPH, "The Dell," Ballycarney Ferns, County Wexford.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1882 GRESSWELL, DAN ASTLEY, M.B., Melbourne, Victoria.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley street, Cavendish square.

1889 GRIFFITHS, JOSEPH, M.B., C.M., 16, Panton street, Cambridge.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, 15, Johnstown street, Bath.

1889 GUBB, ALFRED SAMUEL, 29, Gower street.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

Elected

1885 GULLIVER, GEORGE, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck street.

1883 GUNN, ROBERT MARCUS, M.B., Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street; 54, Queen Anne street, Cavendish square.

1886 HABERSHON, SAMUEL HERBERT, M.D., Casualty Physician to St. Bartholomew's Hospital; 70, Brook street, Grosvenor square.

1888 HADDEN, WALTER BAUGH, M.D., Assistant Physician and Demonstrator of Morbid Anatomy at St. Thomas's Hospital; Assistant Physician, Hospital for Sick Children; 21, Welbeck street, Cavendish square.

1885 HAIG, ALEXANDER, M.D., Casualty Physician to St. Bartholomew's Hospital; 30, Welbeck street, Cavendish square. *Trans.* 5.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 8, Sussex gardens, Hyde Park.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square.

1885 HALLIBURTON, WILLIAM DOBINSON, M.D., Professor of Physiology, King's College, London; 25, Maitland Park Villas, Haverstock Hill.

1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.

1889 HANDFIELD-JONES, MONTAGU, M.D., 24, Montagu square.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

Elected

1856 †HARE, CHARLES JOHN, M.D., *Treasurer*, late Professor of Clinical Medicine in University College, London, and Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-90.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-8. *Sci. Com.* 1879. *Trans.* 10.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. *Trans.* 1.

1854 HAVILAND, ALFRED.

1890 HAVILAND, FRANK PAPILLON, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1870 HAWARD, J. WARRINGTON, *Secretary;* Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-90. *Lib. Com.* 1881-4. *Trans.* 2.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1885 HAWKINS, FRANCIS HENRY, M.B., Physician to St. George's and St. James's Dispensary and to the North London Hospital for Consumption; 59, Wimpole street, Cavendish square.

1848 †HAWKSLEY, THOMAS, M.D., 11, Albert Mansions, Victoria street, and Beomands, Chertsey, Surrey.

Elected

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY.

1848 HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, *Trustee*, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1843 HEWETT, SIR PRESCOTT GARDNER, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Foreign Associate of the "Académie de Médecine," and Corresponding Member of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1887 HEWITT, FREDERIC WILLIAM, M.D., 10, George street, Hanover square.

Elected

1855 †HEWITT, W. M. GRAILY, M.D., Emeritus Professor of Midwifery in University College, London, and Consulting Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-88. *Lib. Com.* 1868, 1874.

1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.

1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 HILL, M. BERKELEY, M.B., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. *Referee*, 1888. C. 1878-9. S. 1881-4. V.P. 1885-6. *Trans.* 1.

1843 †HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 HOLLAND, PHILIP ALEXANDER, M.A.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

1856 †HOLMES, TIMOTHY, M.A., *President*, Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.

1846 †HOLTHOUSE, CARSTEN, 1, Bath terrace, Richmond. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.

Elected

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the North-West London Hospital; Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; Superintendent of the Brown Institution, Wandsworth road; 80, Park street, Grosvenor Square. *Trans.* 1.

1865 HOWARD, BENJAMIN, M.D. [New York, U.S.] *Trans.* 1.

1881 HOWARD, HENRY, M.B., abroad. [6, The Terrace, Mount Pleasant, Cambridge.]

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-8. *Trans.* 2.

1886 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, Surgical Registrar, Middlesex Hospital; Warden of the College.

1884 HUGGARD, WILLIAM R., M.D. [Place de la Synagogue, 2, Genève.]

1857 †HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-90. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 9.

1889 HUMPHERY, FRANCIS WILLIAM, M.A., M.B., 63, Prince's gate, Hyde park.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 8.

1882 HUMPHRY, LAURENCE, M.B., 3, Trinity street, Cambridge.

1889 HUNTER, WILLIAM, M.D., 61, Wimpole street, Cavendish square.

Elected

1873 HUNTER, SIR W. GUYER, M.D., M.P., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 HUSSEY, EDWARD LAW, Consulting Surgeon to the Oxford County Lunatic Asylum and the Warneford Asylum; 24, Winchester Road, Oxford. *Trans.* 1.

1856 †HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-8. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 16, Finsbury circus.

1820 HUTCHINSON, WILLIAM, M.D.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D.

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.

1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., M.B., M.S., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Trans.* 1.

1825 JAMES, JOHN B., M.D.

Elected

1883 *JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.G.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 JENNINGS, CHARLES EGERTON, M.S., M.B., 15, Upper Brook street, Grosvenor square.

1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 JESSOP, WALTER H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib.Com.* 1860-1. *Trans.* 10. *Pro.* 1.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

1889 JOHNSON, HAROLD J., Senior Assistant, Gloucester County Asylum.

1889 JOHNSON, RAYMOND, M.B., B.S., 123, Gower street.

Elected

1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1887 JONES, HENRY LEWIS, M.D., Casualty Physician to St. Bartholomew's Hospital; 6, West street, Finsbury Circus.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1881 JULER, HENRY EDWARD, Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1816 KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1890 KERR, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

1884 KESER, JEAN SAMUEL, M.D., Surgeon to the French Hospital, Leicester place; 11, Harley street, Cavendish square.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

Elected

1857 †KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater. C. 1890.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 4.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and Consulting Surgeon to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., Lecturer on Physiology, St. Bartholomew's Hospital; 19, Earl's Court square.

1883 KNAPTON, GEORGE, 4, Clivedon place, Eaton square.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Demonstrator of Anatomy at St. George's Hospital; 22, Hill street, Knightsbridge.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; St. Mary's, Madeley road, Ealing. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 3.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885-8. *Lib. Com.* 1879-80, 1888. *Trans.* 2.

Elected

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1890 LAWRENCE, HENRY CRIPPS, 12, Sussex gardens, Hyde Park.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

1890 *LAWRIE, EDWARD, M.B., Indian Medical Department; Hyderabad, Deccan.

1884 LAWSON, GEORGE, Surgeon-Oculist to H.M. the Queen; Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., Melbourne, Victoria, Australia.

1886 *LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther street, Carlisle.

1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1884 LEE, ROBERT JAMES, M.D., 6, Savile row.

1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.

1869 LEGG, JOHN WICKHAM, M.D., C. 1886. *Referee*, 1882-5. *Lab. Com.* 1878-85. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

Elected

1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Assistant Obstetric Physician to the London Hospital and Physician to Out-patients of Queen Charlotte's Lying-in Hospital; 60, Wimpole street, Cavendish square.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's Park.

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1889 *LITTLE, JAMES, M.D., Physician to the Adelaide Hospital; 14, Stephen's Green North, Dublin.

1889 LITTLE, JOHN FLETCHER, M.B., 60, Welbeck street, Cavendish square.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; 19, Upper Berkeley street. *Trans.* 1.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Foreign Associate "Académie de Médecine;" Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

Elected

1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1888 LUFF, ARTHUR PEARSON, M.B., B.Sc., 35, Westbourne terrace, Hyde Park.

1883 LUND, EDWARD, Professor of Surgery, and Member of Senate, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1887 LUSH, PERCY J. F., M.B., 8, Fitzjohn's avenue, South Hampstead.

1857 LYON, FELIX WILLIAM, M.D., 7, South Charlotte street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

1889 MACALISTER, DONALD, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

1873 †MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital and Lecturer on Physiology at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5.

1867 MAC CORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Trans.* 1.

1887 MACDONALD, GEORGE CHILDS, M.D.

1880 MACFARLANE, ALEXANDER WILLIAM, M.D., Examiner in Medical Jurisprudence, University of Glasgow; 6, Manchester Square.

1866 MACGOWAN, ALEXANDER THORBURN, M.D., Vyvyan House, Clifton, near Bristol.

Elected

1880 McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., LL.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Trans.* 1.

1885 MACKERN, JOHN, M.D., St. Germain's Lodge, Shooter's Hill road, Blackheath.

1876 MACKEY, EDWARD, M.D., Assistant Physician to the Sussex County Hospital; 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1889 MACLEHOSE, NORMAN MACMILLAN, M.B., C.M., 24, Devonshire street, Portland place.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. *Referee*, 1884-8. *Lib. Com.* 1886-8.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

Elected

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., 4, Seymour street, Portman square.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and to the Evelina Hospital for Children; 2, Queen street, May Fair. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., Surgeon in charge of Out-Patients, Samaritan Free Hospital; 24, Bryanston street, Portman square. *Trans.* 1.

1888 MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park, and Athenæum Club, Pall Mall. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. *Lib. Com.* 1880-1. *Trans.* 4.

1851 †MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 92, Cheyne walk, Chelsea. C. 1866. V.P. 1875-6. P. 1882-3. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

1884 MARTIN, SIDNEY HARRIS COX, M.D.; 10, Mansfield street, Portland place.

1883 MAUDSLEY, HENRY CARR, M.D., 11, Spring street, Melbourne, Victoria.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square.

Elected

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square. *Trans.* 1.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1885 MILLICAN, KENNETH WILLIAM, B.A.

1882 MILLS, JOSEPH, 28, Queen Anne street, Cavendish square.

1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Beaumont Lodge, Worple road, Wimbledon.

1883 MONEY, ANGEL, M.D., Assistant Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond Street, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 24 Harley street, Cavendish square. *Trans.* 4.

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College, and Lecturer on Pathology at, St. Bartholomew's Hospital; The Warden's House, St. Bartholomew's Hospital. *Referee*, 1886-8.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 1.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. C. 1888-9. *Referee*, 1882-7. *Trans.* 10.

Elected

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889.

1885 MOTT, FREDERICK WALKER, M.D., Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton road, Harrow.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Royal Hospital for Incurables; 40, Finsbury square.

1888 †MURRAY, HUBERT MONTAGUE, M.D., 27, Savile row, Burlington gardens.

1873 MURRAY, J. IVOR, M.D., F.R.S.Ed. 24, Huntriss row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Sci. Com.* 1889. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., 9, Lower Berkeley street, Portman square.

1889 NAPIER, FRANCIS HORATIO, M.B., 31, Lower Seymour street, Portman square.

1881 NALL, SAMUEL, M.B., Disley, Stockport, Cheshire.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1889 NEVINS, ARTHUR EDWARD, Eastwood place, Hanley, Staffordshire.

Elected

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Trenanen, Newquay, Cornwall.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; Greenhill Park, Harlesden.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

Elected

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 12, Eldon square, Newcastle-on-Tyne. *Trans.* 1.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889. *Referee*, 1884-8. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 2.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, May Fair.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890. *Referee*, 1884-8. *Lib. Com.* 1886-8. *Trans.* 4.

1887 PAGET, CHARLES EDWARD, North Bentcliffe, Eccles, Lancashire.

Elected

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 PAGET, STEPHEN, 57, Wimpole street, Cavendish square.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; The Old Residence, Ripon, Yorkshire.

1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. C. 1888-9. *Lib. Com.* 1885-7. *Trans.* 4.

1885 PARKER, RUSHTON, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1889 PARSONS, J. INGLIS, M.D., 3, Queen street, May Fair.

1883 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen street, May Fair.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

Elected

1856 PEIRCE, RICHARD KING, Laggan House, Maidenhead.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 11, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Thema, Worcester.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 24, Clarges street, Piccadilly.

1890 PERRY, EDWIN COOPER, M.D., The College, Guy's Hospital.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., F.R.S.Ed., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.

1888 PHILLIPS, JOHN, M.B., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square.

1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients at St. Mary's Hospital, and Assistant Physician to the London Fever Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

Elected

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark.

1889 PITTS, BERNARD, M.B., M.C., 31, Harley street, Cavendish square.

1885 POLAND, JOHN, Demonstrator of Anatomy, Guy's Hospital; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, Assistant Surgeon and Surgical Registrar to University College Hospital, Surgeon to the North Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. C. 1890. *Referee*, 1887-8. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen, Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 62, Wimpole street, Cavendish square. S. (Oct.), 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.* 3.

Elected

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Practical Surgery at St. Bartholomew's Hospital; Surgeon to Out-patients at Victoria Hospital for Children; 26, Bloomsbury square.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 †PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to, and Physician in Charge of Skin Department at, the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton street, Berkeley square. *Trans.* 1.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital and to the Victoria Hospital for Children; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley street, Cavendish square. *Lib. Com.* 1887-8.

1850 †QUAIN, RICHARD, M.D., LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

Elected

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1890 RANSOM, WILLIAM BRAMWELL, M.D., The Pavement, Nottingham.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1858 †REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1882 REID, JAMES, M.D., C.B., Resident Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

1855 †REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1887 RICHARDSON, GILBERT, M.D., Hawthorne House, Putney.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 6.

1889 RIVERS, W. H. RIVERS, M.D., National Hospital, Queen Square.

1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 22, Finsbury square. C. 1885-6. *Trans.* 4.

Elected

1871 *ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary, Physician to St. Mary's Hospital, Manchester; 11, St. John street, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square.

1889 ROBERTS, HUGH LESLIE, M.B., C.M., 31, Rodney street, Liverpool.

1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

1888 *ROBINSON, FREDERICK WILLIAM, M.B., C.M., Huddersfield.

1889 ROBSON, ARTHUR WILLIAM MAYO, Hillary place, Leeds. *Trans.* 1.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

1857 †ROSE, HENRY COOPER, M.D., F.L.S., Consulting Surgeon to the Hampstead Dispensary; 53, Rosslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Surgery at King's College, Surgeon to King's College Hospital and to the Royal Free Hospital; 17, Harley street, Cavendish square.

1889 ROSS, DANIEL MCCLURE, 54, Upper Berkeley street, Portman square.

Elected

1888 ROUGHTON, EDMUND WILKINSON, M.B., B.S., 60, Gloucester place, Portman square. *Trans.* 1.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Obstetric Physician to the Charing Cross Hospital; 14A, Manchester square.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Senior Visiting Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital and Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 †SANDERSON, JOHN BURDON, M.D., LL.D., D.C.L. Durham, F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

Elected

1879 SANGSTER, ALFRED, B.A., M.B., Physician to the Skin Department, and Demonstrator of Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1869 SANSOM, ARTHUR ERNEST, M.D., Senior Physician to the North-Eastern Hospital for Children; Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. C. 1887-8. *Trans.* 2.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; 83A, Edmund street, Birmingham.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta street, Cavendish square.

1859 SAVORY, SIR WILLIAM SCOVELL, Bart., F.R.S., Surgeon Extraordinary to H.M. the Queen, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888.

1887 SCOTT, HARRY, M.D., 28, Great Smith street, Westminster.

1861 *SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

Elected

1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Consulting Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR JOHN, M.D., Assistant Physician, Joint Lecturer on Pathology, and Demonstrator of Morbid Anatomy, to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician, Medical Registrar, and Demonstrator of Practical Medicine at Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street. *Trans.* 1.

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., 123, Pall Mall.

1848 †SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

Elected

1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of out-patients, St. Mary's Hospital; Assistant Surgeon, Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square.

1842 †SIMON, Sir JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1890 SMALE, MORTON, 22A, Cavendish square.

1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1866 SMITH, HEYWOOD, M.A. M.D., 18, Harley street, Cavendish square.

1886 SMITH, HOWARD LYON.

1885 SMITH, JAMES GREIG, M.B., C.M., F.R.S.Ed., Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol.

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1889 SMITH, ROBERT PERCY, M.D., B.S., Bethlem Royal Hospital.

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

Elected

1863 SMITH, THOMAS, *Vice-President*, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Ed., Professor of Forensic Medicine at King's College, London; 74, Great Russell Street, Bloomsbury. *Trans.* 1.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK ROBERT, M.D., Consulting Physician to St. Alban's Hospital, Harpenden, St. Alban's.

1889 SPENCER, HERBERT R., M.D., B.S., 10, Mansfield street, Portland place.

887 SPENCER, WALTER GEORGE, M.B., Assistant Surgeon to the Westminster Hospital; 94, Wimpole street, Cavendish square.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 6A, Bedford square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 53, Harley street, Cavendish square. *Trans.* 1.

Elected

1882 STEAVENSON, WILLIAM EDWARD, M.D., Electrician to St. Bartholomew's Hospital; Physician to the Alexandra Hospital for Children; 15, Mansfield street, Portland place.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board, Whitehall; Mitcham House, Mitcham, Surrey.

1884 STEWART, EDWARD, M.D., 8, Upper Wimpole street, Cavendish square.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

1856 †STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum, University College, London; 62, Welbeck street, Cavendish square.

1843 STORKS, ROBERT REEVE.

1871 STRONG, HENRY JOHN, M.D., Surgeon to the Croydon General Hospital; Whitgift House, George street, Croydon.

1863 †STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1882-8.

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury square. *Referee*, 1888. *Trans.* 1.

Elected

1883 SUTTON, JOHN BLAND, Assistant Surgeon, Lecturer on Comparative Anatomy, and Senior Demonstrator of Anatomy to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 5.

1890 SYERS, HENRY WALTER, M.D., 3, Devonshire street, Portland place.

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth street, Portland place.

1890 SYMPSON, E. MANSEL, M.A., M.B., B.C., 5, James street, Lincoln.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WABEN, Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, to the North Eastern Hospital for Children, and to the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Secretary;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-90. *Sci. Com.* 1889. *Referee,* 1887-8. *Trans.* 1.

1890 TAYLOR, SEYMOUR, M.D., 16, Seymour street, Portman square.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's Wood.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's. C. 1888-9.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

Elected

1862 THOMPSON, EDMUND SYMES, M.D., Senior Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889. *Referee*, 1876-7. *Trans.* 1.

1852 †THOMPSON, SIR HENRY, *Vice-President*, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., late Senior Consulting Surgeon to Peterbro' Hospital, and Medical Officer of Health for Peterbro'; 1, Palace court, Notting Hill gate.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Lib. Com.* 1886-8. *Trans.* 5.

1885 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., 28, Weymouth street, Portland place.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

Elected

1882 TOOTH, HOWARD HENRY, M.D., Assistant Medical Tutor St. Bartholomew's Hospital; 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., 1, Grosvenor square, Southampton.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Sci. Com.* 1889. *Trans.* 5.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1889 TURNBULL, GEORGE LINDSAY, M.B., 121, Ladbroke grove.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., Vernon House, Ryde, Isle of Wight.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green street, Park lane.

1888 TYLDEN, HENRY JOHN, M.B., 38, Harewood square.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician for the Diseases of Women, West London Hospital; 122, Harley street, Cavendish square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

Elected

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and to the French Hospital, Leicester place; 19A, Hanover square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., 67, Grosvenor street, Grosvenor square.

1864 WAITE, CHARLES DERBY, M.B., Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.

1884 WAKLEY, Thomas, jun., 5, Queen's Gate, South Kensington.

1868 *WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1883 WALLER, AUGUSTUS, M.D., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., 18, St. James's street.

1867 WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. C. 1888-9. *Lib. Com.* 1882-5. *Trans.* 5.

1852 †WALSHE, WALTER HAYLE, M.D., LL.D.Edin., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption and to University College Hospital; 41, Hyde Park square. C. 1872. *Trans.* 1.

Elected

1883 *WALTERS, JAMES HOPKINS, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1886 WARD, ALLAN OGIER, M.D., 1, Brook place, Lower Tottenham.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton Gardens Maida vale. *Referee*, 1881-5.

1877 WARNER, FRANCIS, M.D., Assistant Physician and Lecturer on Botany to the London Hospital; 5, Prince of Wales Terrace, Kensington Palace. *Trans.* 1.

1889 WASHBOURN, JOHN WYCHENFORD, M.D, Assistant Physician to Guy's Hospital; 14, St. Thomas's street.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., Physician in Charge of the Electro-therapeutical Department at St. Mary's Hospital; 30, Welbeck street, Cavendish square.

1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

Elected

1878 WEISS, HUBERT FOVEAUX, Assistant Surgeon to the West London Hospital; 11, Hanover square.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1854 †WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Corresponding Member, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Foreign Associate of the Academy of Medicine of Paris; 55, Harley street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Trans.* 4.

1888 WETHERED, FRANK JOSEPH, M.B., 34, Queen Anne street, Cavendish square.

1882 WHARRY, CHARLES JOHN, M.D.

1881 WHARRY, ROBERT, M.D., Physician to the Westminster Dispensary; 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

Elected

1881 WHITE, WILLIAM HALE, M.D., Senior Assistant Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888. *Trans.* 2.

1890 WHITE-COOPER, G. O., M.B., 5, Cranley gardens, Brompton.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, and to the Manchester and Salford Lock and Skin Hospital; 499, Oxford road, Manchester. *Trans.* 1.

1885 *WHITLA, WILLIAM, M.D., Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Surgeon to the Westminster General Dispensary; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham park, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught, and to H.R.H. the Duke of Edinburgh; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1890 WILLCOCKS, FREDERICK, M.D., 14, Mandeville street, Manchester square.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890. *Reverse*, 1882-8. *Trans.* 2.

Elected

1887 WILLETT, EDGAR WILLIAM, M.B., 60, Welbeck street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1888 WILLIAMS, CAMPBELL, 62, Welbeck street, Cavendish square.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888. *Lib. Com.* 1880-3. *Trans.* 5.

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; 25, Old Burlington street.

1872 WILLIAMS, JOHN, M.D., Physician Accoucheur to H.R.H. the Princess Beatrice; Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. *Referee*, 1878-88. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Linden House, Bertie road, Leamington.

1890 WILLS, WILLIAM ALFRED, M.B., 52, Davies street, Berkeley square.

1887 WILSON, ARTHUR HERVEY, M.D., 504, Broadway, Boston, U.S.A.

1889 WILSON, JOHN HENRY PARKER, H.M.'s Military Prison, The Avenue, Brixton Hill.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1889 WISE, A. TUCKER, M.D., Kursaal de la Maloja.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn-Banbury.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

Elected

1885 WOLFENDEN, RICHARD NORRIS, M.D., Assistant Physician to the North-West London Hospital; 19, Upper Wimpole street.

1851 †WOOD, JOHN, F.R.S., 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-88. *Lib. Com.* 1866. *Trans.* 3.

1887 WOOD, THOMAS OUTTERSON, M.D., 40, Margaret street, Cavendish square.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., The Priory, Roehampton.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Hon. Secretaries before the 1st of September in each year, in order that the List may be made as correct as possible.]

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1833 Thomas A. Barker, M.D.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
Sir William Bowman, Bart., F.R.S.
Paul Jackson.
1842 Charles West, M.D.
Sir John Simon, K.C.B., F.R.S.
John Erichsen F.R.S.
Sir Oscar M. P. Clayton, C.M.G.
1843 Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Luther Holden.
Edward Newton.
1844 William Wegg, M.D.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
Edward U. Berry.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 George Johnson, M.D., F.R.S.
1848 Sir Edward H. Sieveking, M.D.
Edward Ballard, M.D., F.R.S.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
1848 John Clarke, M.D.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
1850 Richard Quain, M.D., F.R.S
1851 Sir Wm Jenner, Bt., M.D., F.R.S.
John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
1852 Walter H. Walshe, M.D.
William Adams.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Sir Alfred Baring Garrod, M.D., F.R.S.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
William Marcet, M.D., F.R.S.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Hermann Weber, M.D.
John Whitaker Hulke, F.R.S.
John Morgan.

1857 Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
Sir William Scovell Savory, Bart., F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D., F.R.S.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
Robert Farquharson, M.D., M.P.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley M.D.
Thomas William Nunn.
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Regulations relative to the publication of the 'Proceedings of the Society.'

The 'Proceedings' are issued after each Meeting.

They are sent, postage free, to every Fellow of the Society who, in writing, expresses a wish to receive them.

They may be had by others at the Society's House, on payment (in advance) of an annual subscription of five shillings and eightpence, which may be sent either by post-office order or in postage-stamps;—this will include the expense of conveyance by post to any place within the Postal Union. For places beyond the Postal Union special arrangements must be made.

A notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretaries whether they wish them to appear in the 'Proceedings' *only*, or in the 'Proceedings' and 'Transactions;' and in all cases they must furnish an Abstract of the communication.

The Abstracts of the papers read are sent to the Journals as heretofore.

ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

ADDRESS

ON THE OCCASION OF THE

FIRST MEETING IN THE NEW HOUSE OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY, No. 20, HANOVER SQUARE, ON TUESDAY, OCTOBER 22, 1889.

BY

SIR EDWARD H. SIEVEKING, M.D., LL.D., F.R.C.P.,
PRESIDENT.

FELLOWS OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY

Welcome to our New Home!

THE new phase which our Society is this day entering upon, not only appears to justify, but to call for a brief consideration of the admirable work performed by our founders and predecessors, as well as of the duties which we have undertaken to medical science and to humanity. The small beginings which ushered in the first formation of the Society have been nurtured and fostered, until greater expansion became imperative; and we have now entered upon what, as far as we can see into the future, promises to remain the home of the Royal Medical and Chirurgical Society for many future generations. The wishes which are now, thanks to the admirable management of the Building Committee and the energy of the Fellows, on the eve of fulfilment, have for some years been in the hearts and mouths

of all our supporters. We have long felt cramped in our Berners Street residence, where at last, all the ingenuity that could be brought to bear, failed to make room for the work that we were called upon to do, and to house the ever-increasing library, our most precious heir-loom and possession.

No history of our Society is at present available. The following notices therefore, gathered together from authentic records, may not be without interest; and though necessarily meagre and disjointed, may prove useful to the future writer possessed of the ability and leisure to exhibit the true development of the Society, as displayed by the scientific growth that has characterised it, and by the fostering care with which it has watched over medicine and surgery.

On May 22nd, 1805, an inaugural meeting was held at Freemasons' Tavern,[1] Dr. Saunders, F.R.S., F.R.C.P., in the chair, at which it was determined:

1. That a Society comprehending the several branches of the Medical Profession be established in London, for the purpose of conversation on professional subjects, for the reception of communications, and for the formation of a library.

2. That this Society be denominated the Medical and Chirurgical Society of London.

3. That its meetings be held in some central situation.

4. That its affairs be conducted by a President, four Vice-Presidents, a Treasurer, three Secretaries (one of whom shall be Foreign Secretary) and a certain number of members, who together shall constitute a Council and shall be elected annually.

5. That no gentleman shall be eligible to the office of President or Vice-President for more than two years in succession.

6. That a certain number of the Council go out annually.

7. That six guineas be the sum subscribed on admis-

[1] This is still in existence in Great Queen Street, Lincoln's Inn Fields, though much altered. I am, however, informed by the manager that some of the old rooms belonging to the original building are in existence.

sion, and that three guineas annually be subscribed for the use of the Society.

8. That after the organisation of the Society all admission into it be by ballot, and that no person be declared elected unless he have in his favour at least three fourths of the numbers voting.

These are the main points determined upon at this preliminary meeting, and their wisdom cannot be better demonstrated than by the fact that they continue to rule our conduct. Twenty-six gentlemen were at once inscribed as regular members, and Dr. Yelloly was requested to act as Secretary of the Committee nominated for the purpose of preparing a plan of further operations. This Committee consisted of eighteen of the original members.

The first meeting of the Society was held at No. 2, Verulam Buildings, Gray's Inn, in December, 1805, but it was not till 1809 that the first volume of our 'Transactions' was published, a publication which, as we believe it to redound to the honour of the Society, we hope has been profitable as well as honourable to the ancient and respected firm of publishers,[1] who from the commencement have behaved to the Society with liberality.

Before going any further it may be interesting to you to know the intellectual basis upon which our Society was founded. The members of the first Council, which held the reins of office from 1805 to 1807, were all men who have contributed largely to the advancement of medical science, and no future history of our profession will be complete without an admiring record of much of the work performed by them. Many of their names are even yet household words among us. The following may be regarded as the founders as they were the first rulers of the Medical and Chirurgical Society[2]:—William Saunders, M.D., F.R.S., President; John Abernethy, Esq., F.R.S., Vice-President; Charles Rochemont Aikin, Esq.,

[1] Messrs. Longman and Co.

[2] The sequence of the names is that given in the first volume of the 'Transactions.'

Secretary; William Babington, M.D., F.R.S., Vice-President; Matthew Baillie, M.D., F.R.S.; Thomas Bateman, M.D., F.R.S.; Gilbert Blaine, M.D., F.R.S.; Sir William Blizzard, F.R.S., Vice-President; Astley Cooper, Esq., F.R.S., Treasurer; James Curry, M.D., F.R.S.; Sir Walter Farquhar, M.D.; John Heaviside, Esq., F.R.S.; Alexander Marcet, M.D., F.R.S., Foreign Secretary; David Pitcairn, M.D., F.R.S.; Henery Revell Reynolds, M.D., F.R.S.; H. Leigh Thomas, Esq., F.R.S.; James Wilson, Esq., F.R.S., and John Yelloly, M.D., Secretary.

The only material difference that suggests itself between this list and the lists that you are familiar with, is the elimination of the Foreign Secretary. The greater intercourse which has existed between this country and the Continent has doubtless increased the accomplishments of our more recent secretaries to such an extent that they have not for many years required the aid of a special official to enable them to carry on a foreign correspondence.

The first volume of the 'Transactions' appeared in 1809, and had the distinction of being reprinted for a third time in 1815. It opens with a paper by Astley Cooper on a case of Aneurism of the Carotid Artery, followed by one of Dr. Stanger on Violent and Obstinate Cough; on the Treatment of Whooping-Cough by Dr. Pearson; a paper by Dr. Bostock, of Liverpool, on the Gelatine of the Blood; one by Thompson Forster, a case of Lithotomy; and by Dr. Marcet on the Effects of Large Doses of Laudanum and their Remedies. These are dated 1806. The following belong to the years 1807 and 1808:—An Account of a Peculiar Disease of the Heart by David Dundas, Sergeant-Surgeon; a case of Exposure to the Vapour of Burning Charcoal by Dr. Babington; on Gouty Concretions by Mr. Moore; a case of Artificial Dilatation of the Female Urethra by Mr. Thomas; a case of Hydrophobia by Dr. Marcet; three cases of Sudden Death, with Post-mortem, by M. Cheva-

lier; a case of Intussusception by Mr. Blizzard, and a description of Two Muscles surrounding the Membranous portion of the Urethra by Mr. J. Wilson. This is followed by a case of Tumour of the Brain by Dr. Yelloly, and a second case of Carotid Aneurysm by Astley Cooper; then we come upon a case of a Fœtus found in the Abdomen of a Boy by Geo. Will. Young; two cases of Smallpox Infection communicated to the Fœtus in Utero under Peculiar Circumstances by Dr. Jenner, and an historical account, by Dr. White, of Philip Howorth, a boy in whom signs of Puberty commenced at an Early Age, which was communicated by Dr. Yelloly, conclude this catalogue. A list of works given by Baillie, Hunter, Astley Cooper, and others, and which formed the nucleus of the magnificent library we now possess, ends this first volume of the 'Medical and Chirurgical Transactions.'

From this time forth the Society's 'Transactions' have appeared, with very few interruptions, annually up to the present day, and have contributed in no small degree to give an excellent stamp to its fellowship and to British medicine at large. The Society steadily increased in numbers and in influence, but it was evidently cramped at first by the *res angusta domi*, for we find that in these early days the Council resolved that Mr. Nichols (Clerk) be allowed to occupy "the library when it is not otherwise wanted, and to procure a press-bedstead at the Society's expense for his accommodation, to stand in the further corner of the meeting room."

In the year 1810 No 3, Lincoln's Inn Fields became the home of the Society, which it occupied in conjuction with the Geological Society, from whose President, on the security of three Fellows, the Medical and Chirurgical Society borrowed £200. The two societies did not separate till 1816. Nos. 30 and 57, Lincoln's Inn Fields[1] were

[1] It appears that No. 57, Lincoln's Inn Fields was taken conjointly with the Astronomical Society from Midsummer Day, 1821, on a 7, 14, or 21 years' lease, terminable at the option of either party on giving six months' notice.

temporarily our home until, in 1834, for a long series of years the property of the Society was moved to No. 53, Berners Street. The first meeting assembled in this locality on February 3rd, 1835, the last on June 11th, 1889.[1] Although difficulties occasionally surrounded the Society, especially in the early part of its existence, nothing appears materially to have checked its work and steady growth. For the first time, in 1812, we find the Society associating with itself Foreign Honorary Members; their names are still mentioned with veneration: Blumenbach, Cuvier, Rush (of Pennsylvania), Sömmering, Corvisart, Odier, Scarpa, and Vieussieux (of Geneva).[2] It may be mentioned that the year 1834, during which the removal from Lincoln's Inn was effected, and the cost of which was defrayed by voluntary subscriptions[3] of the Fellows, was marked by the absence of a volume of the 'Transactions,' owing to the confusion necessarily arising from the migration. At this time the agitation for a Royal Charter was successful, and since then we have been incorporated as the Royal Medical and Chirurgical Society of London,[4] of which the Sovereign (in the first instance, William the Fourth) is Patron, by which name we shall "have perpetual succession and a Common Seal," with such other rights and privileges as belong to any other body, politic and corporate, in "our United Kingdom of Great Britain." It was not without a struggle that this Charter had been obtained; the Society had made great efforts during the years 1812, 1813, and 1814 to achieve this object of their ambition. In the month

[1] The last meeting of Council was held at 53, Berners Street, in Mr. MacAlister's rooms on Oct. 15th, 1889.

[2] The first proposal paper, still in our possession, is in manuscript. It was that of Thomas Young, M.D., F.R.S., Physician to St. George's Hospital, and is signed by Alex. Marcet, Henry Halford, Robert Bree, J. Yelloly, and P. M. Roget. He was proposed Nov. 12th, 1812, and balloted for on Jan. 5th, 1813.

[3] They amounted to £346 9*s*.

[4] This charter was granted to Dr. Elliotson, Sir Astley Cooper, Bart., and Dr. Yellolp.

of February, 1812, "in consequence of the gracious reception accorded by H.R.H. the Prince Regent to an application of Sir Henry Halford," who was at that time President of the Medical and Chirurgical Society, a petition for a Charter was sanctioned by the Society; after having been signed by Sir H. Halford, President, Drs. Saunders and Baillie, past Presidents, by Sir Walter Farquhar, Drs. Marcet and Yelloly, and Messrs. Clive, Abernethy, and Cooper, the Trustees of the Society, it was laid before H.R.H. the Prince Regent in Council. I regret to say that the Royal College of Physicians, under the Presidency of Sir Francis Milman, objected to the grant, stating in their counter-petition that they would be materially aggreived by the grant of a Royal Charter of Incorporation to the Medical and Chirurgical Society. The grounds upon which the College based its opposition would be unintelligible to the present generation, but as an item in the history of British medicine, I think it right to place before you some of the arguments employed on behalf of the College. It was said by its defenders that "by certain Regulations or Bye-Laws of the said College, any tract or treatise on medical subjects, written by any Fellow or Candidate of the said College, or by any person licensed by the said College to practise physic, may be read at certain meetings of the said College, and if approved of, in manner as by such Regulations and Bye-Laws is required, will be directed to be printed at the expense of the said College;" and again, "that the establishment of such Society by Royal Charter will be the means of depriving the College of Physicians of such tracts upon medical subjects as shall be written by those members of the College who shall likewise be members of the Medical and Chirurgical Society."

The Attorney-General and Solicitor-General took the part of the College of Physicians, and in spite of a long correspondence, the Privy Council agreed, at the Court at Carlton House, the 19th March, 1814, present H.R.H. the Prince Regent in Council:—"That they do not see suffi-

cient grounds for recommending the grant of a charter to the Medical and Chirurgical Society."

Of this report the Prince Regent was pleased to approve, and the charter accordingly was not granted.

Let us hope that if Sir Francis Milman and his coadjutors were alive now, they would, in the favourite language of the College in those days, repentantly join us in exclaming, "O cœca mens mortalium!"

Our Society seems to have for some time been exercised by the desire of bestowing prizes upon distinguished Fellows; we find references to this matter in 1822 and again in 1837, but nothing was ever done in this direction, until the foundation, by voluntary subscription, of the Marshall Hall Prize, in 1872, as a memorial to a great man; and which has been since awarded to three distinguished workers in the same field as that in which he laboured so successfully: Drs. Hughlings Jackson, Ferrier, and Gaskell.—The whole question of prize-giving is one that may be discussed from various points of view, but if there is a difference of opinion in regard to some aspects of the question, it can scarcely be doubted that it is a high function for a society like ours to perform, to award a tangible proof of its appreciation of good work done in the cause of science under the restrictions such as those surrounding the Marshall Hall Prize. You will remember that this is a quinquennial prize bestowed for work not necessarily done in connection with our Society. This was not the former object of the Society, for we read in the 'Transactions' of 1823 the resolution, "that the Council shall adjudge out of the funds of the Society a prize to the author of the paper that shall appear to them most deserving of that honour, amongst those that shall have been read to the Society during the session."

It is to be assumed that the Council were satisfied that they had sufficient pecuniary means at their disposal or they would not have made such a proposition. It is easily intelligible that the great variety of subjects treated by different Fellows, would render it very difficult to judge

of their productions according to any common standard. Although I have no definite data to rely upon, I have little doubt that this was the rock on which the good intentions of the Council were wrecked. Whenever our Treasurers report an ample credit balance, and we are again tempted to establish prizes, let us profit by the experience of the past. There are ample methods of spending our money for the advancement of science, and so long as we appoint good working committees for the special investigation of questions bearing upon medicine and surgery we need never be at a loss as to spending our money for the promotion of the objects defined by our Charter.[1]

Our removal to Berners Street, and our receiving the honour of a Royal Charter of Incorporation, was marked by the termination of the first series of eighteen volumes of our 'Transactions' and by the commencement of the series which is still running on. It will be a question for your consideration whether it may not be well to mark the great event of our emigration to a new home in a similar manner as was done by our predecessors. I, for one, see certain advantages in defining in a society like ours, the footsteps of time; and, as far as my judgment guides me, I do not apprehend any counteracting drawbaks.

When I had the honour of serving the office of Secretary to the Society there was a prolonged agitation on the subject of establishing a fusion of the numerous societies formed for the promotion of distinct branches of the medical profession. I cannot say that I saw my way to removing the various difficulties in the way of the execution of a plan which, theoretically, promised very well. Nor do I think that the difficulties are less now or that there would be a greater prospect of success if the different societies took up the question at the present day. But it does suggest itself to me that as six societies of a scientific character and one with philanthropic purposes, all closely

[1] The Society's Charter states that "the Society was formed for the cultivation and promotion of physic and surgery, and of the branches of science connected with them."

associated with the medical profession, will in future occupy rooms under our roof, there will be many opportunities of carrying out objects that an Academy of Medicine would have in view, but which would be perhaps even more energetically and beneficially realised by the co-operation of Societies that are now distinct in their objects, their means, and their government. There are many questions of medical science and medical government which would be more completely solved and more actively prosecuted by joint committees of the six scientific societies working with the Royal Medical and Chirurgical Society than could be achieved by any one of them carrying on their labours alone.

But a few words more and I will call upon the Secretary to initiate the regular work to which our assemblies are devoted. If we are deeply indebted to our predecessors for what they have done in establishing this Society on a broad and firm basis, we must not forget the debt of gratitude we owe to those of our contemporaries who have made it possible for us to enter into possession of this palatial edifice, a home that Medical Science will claim as its own, we trust, for centuries to come, and where, "widening down from precedent to precedent," true and beneficial knowledge may find expansive and increasing power for good through many future generations of Britons.

Some unavoidable delays have prevented the house being as far advanced towards completion as your Council had reason to expect when the contracts were first signed, but the Building Committee and your Council were equally of opinion that it would be better to assemble even in the present condition of the rooms than to seek a temporary home. It is only right to mention that the Medical Society of London through its President, Dr. Theodore Williams, who is also a distinguished Fellow of this Society, knowing of some difficulties that beset us, offered us the use of their rooms. The fraternal spirit that suggested this offer is an admirable illustration of the good feeling and harmony that pervades the republic of science.

Much, however, has been achieved in the brief space of time that has been at our disposal, and it is only right that you and future Fellows of the Society should know upon whom the chief burden and anxiety has fallen. The members of the Building Committee, to whom the Council, with your sanction, have delegated the great responsibility of carrying out this important work, are Mr. Timothy Holmes (Chairman), Dr. Cheadle, Dr. Gee, Dr. Hare, Dr. Isambard Owen, Mr. Warrington Haward, Mr. R. W. Parker, and Mr. A Willett. Our architect is Mr. Flockhart. I am quite sure that I only echo the opinion of every Fellow of the Society if I couple with this distinguished list of names, that of a gentleman who, though not a member of the Building Committee, has from the first inception of the plan shown an amount of tact, zeal, and ability which has materially lightened the labours of the Committee and Council, I mean our Resident Librarian, Mr. MacAlister.

It now only remains for me to express a hope that I am sure you will all echo, that God may bless the Royal Medical and Chirurgical Society in its new home, and prosper its work, carried on for the advancement of science and for the benefit of our countrymen and of humanity at large.

ANNUAL MEETING OF THE SOCIETY,

SATURDAY, MARCH 1st, 1890.

Sir Edward Henry Sieveking, M.D., LL.D., President, in the Chair.

Frederick Taylor, M.D.,
J. Warrington Haward, F.R.C.S., } Hon. Secs.

Present—101 Fellows.

The President nominated Dr. Stephen Mackenzie and Mr. W. A. Meredith to scrutinise the results of the election of officers and Council for the ensuing year, and declared the ballot open for one hour.

The President then called upon Mr. Warrington Haward (Hon. Sec.) to read the

Report of the President and Council.

The President and Council have this year to present to the Society a more than usually important Report, inasmuch as since the last Annual Meeting a change of residence has been determined upon and carried out.

It had for some time past been felt that the premises in Berners Street occupied by the Society since the year 1834 had become both inconvenient and inadequate. Every year there was increasing difficulty in placing the books of the constantly growing Library. The room used for the Society's meetings, the Council room, and the Committee rooms all contained books; and as the meeting-

room was the only place available for study, its furniture had to be rearranged for every meeting that took place. The small reading-room was very crowded and uncomfortable, and had no sufficient space for the display of the current journals. Moreover, during recent years an increasingly large number of the Fellows of the Society had moved towards the west of London, so that the situation of the Society's house was in this relation far from central.

It had also become apparent that the state of the house necessitated a considerable expenditure upon repairs, besides the alterations needful for providing for the books in a manner which was at the best but inconvenient and temporary, as well as expensive.

All these considerations pointed to the desirableness of seeking other and more commodious premises, in favour of which was also the fact that the lease of the Berners Street house had only sixteen years to run, and was therefore becoming yearly less saleable.

But, on the other hand, there was the great difficulty of finding appropriate rooms for the Society's needs in a suitable locality and at an attainable price, and inquiries made from time to time had only made the difficulty more apparent.

At the beginning of 1889, however, it came to the knowledge of the Council, through the assiduous inquiries of our Resident Librarian, Mr. Mac Alister, that the house No. 20, Hanover Square, had come into the hands of two gentlemen who were about to enter into contracts for a complete reconstruction of the building, but that these gentlemen were open to an offer for the property if made at once and before their proposed contracts were signed.

The house was a freehold, and, with the land attached to it, seemed so admirably suited for the purposes of the Society, that a Special Meeting of the Council was held on February 20th, 1889, to consider the matter, and a Committee was appointed for the purpose (*a*) of obtaining information and reporting to the Council concerning the

exact terms on which the house could be obtained; (*b*) of obtaining a valuation of the property by an expert; (*c*) of preparing a financial statement showing the present and prospective liabilities of the Society.

As the result of their investigations the Committee ascertained—

(1) That No. 20, Hanover Square, was a freehold house on the west side of the square, with a frontage of rather more than fifty feet.

(2) That the house consisted of four floors, containing in all twenty-three rooms, besides a very extensive arched basement; that beyond the house was a garden 50 feet broad and 140 feet long, at the end of which was a nine-stalled stable with coach-house and rooms over, to which was an entrance from Bond Street. The freehold property extended only to about the first 100 feet of the garden, the remaining portion of garden and the stables being held on lease from the Corporation of London, at the annual rent of £5 15*s.*, 37½ years of the lease being unexpired.

(3) That Messrs. Elgood, having valued the property, advised that £22,000 or £23,000 would be a reasonable price for it.

(4) That the lowest sum the owners would accept for the property was £23,000.

This information was laid before the Council in a report of the Committee presented on February 27th, together with a financial statement prepared with the assistance of Mr. Francis Cooper (professional accountant).

With these facts before them, the Council, at a special meeting on February 27th, 1889, after taking all the circumstances into careful consideration, and having regard especially to the exceptional opportunity offered of obtaining suitable *freehold* premises, decided to recommend the purchase of the premises, No. 20, Hanover Square, for a sum not exceeding £23,000.

A Special General Meeting of the Society to consider this recommendation of the Council was held on March 4th,

1889; Sir Edward Sieveking, President, in the Chair, at which the following resolutions were passed:

(1) That the recommendation of the Council to purchase the house, No. 20, Hanover Square, be and is hereby approved by the Fellows of the Royal Medical and Chirurgical Society of London in Special General Meeting assembled, and that steps be immediately taken to carry this recommendation into effect.

(2) That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to do one or all or several of the following acts for and on behalf of the Society, namely:

(i) To acquire the freehold and leasehold property in the premises at No. 20, Hanover Square, by purchase, for a sum not exceeding £23,000, such property to be vested in the Trustees of the Society.

(ii) To lease, sell, or mortgage the leasehold premises now occupied by the Society at No. 53, Berners Street.

(iii) To raise such funds as may be required for the acquisition of the premises in Hanover Square, and for such additions and alterations as may be required—

(*a*) By mortgage of the Society's leasehold and of the property to be acquired.

(*b*) Or by the issue of bonds among the Fellows of the Society.

(*c*) Or by such other means as may seem to the Council most advantageous to the interests of the Society.

(iv) To do all such acts and employ such persons as are necessary or advisable for the carrying out of these purposes.

(3) That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to instruct the Trustees of the Society to sell out the securities now vested in their names, and to pay the proceeds of such sale to the Treasurers.

(4) That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to appoint a Building Committee to complete the purchase

of the new premises, and under the direction of the Council to carry out such alterations and repairs as are required therein, and that such Committee consist of the following gentlemen: Dr. Cheadle, Dr. Gee, Dr. Hare, Dr. Isambard Owen, Mr. Timothy Holmes, Mr. Alfred Willett, Mr. R. W. Parker, and Mr. Warrington Haward, and that the Council have power to add to to the number of the Committee if they think it necessary.

To this Committee was subsequently added Mr. Clinton T. Dent, to whom the Society is greatly indebted for his valuable aid in regard to the electric lighting.

These resolutions were confirmed (in accordance with Bye-Laws, Chap. XVIII, Sec. 5) at a Special General Meeting of the Society held on March 11th, 1889.

The funds for carrying out these purposes were soon subscribed by 120 Fellows of the Society, and on March 20th and April 16th, 1889, meetings of the subscribers to the Debenture Fund were held, at which it was determined—

(1) That Dr. Thomas Barlow, Dr. Gee, Dr. C. Theodore Williams, and Mr. Warrington Haward be appointed Trustees, to whom the Society's freehold and leasehold property is to be mortgaged for the security of the Debenture holders.

(2) That in addition to this security, Debentures should be issued to the Subscribing Fellows, acknowledging the Society's indebtedness to the extent of the several amounts advanced.

(3) That the Loan be issued in £50 Debentures, to be redeemed at the end of fifty years; that the interest at 4 per cent. be paid by half-yearly Coupons to Bearer; that the Society reserves to itself the right to pay off any Debentures at any time on three months' notice being given; that for five years no repayment of principal should be guaranteed, but that after five years from the date of the Loan the formation of a Sinking Fund should be commenced, to which yearly additions should be made for the ultimate repayment of the Loan.

(4) That the Debentures be issued to, and in favour of, the Fellows of the Society contributing to the Loan, and that the form of Debenture and Trust Deed be such as to provide that Debentures shall not, without consent of the Council, be assigned to any one not being a Fellow, except in case of the death of a registered holder, in which case the right thereto shall be vested in his executor or administrator for the purpose only of assigning to and vesting the same in any specific Legatee or Legatees thereof or (if there shall be no such Legatee) in some person being a Fellow of the Society, unless in the meantime the same shall be drawn for payment.

A Committee of Subscribers to the Loan, consisting of Sir Andrew Clark, Dr. Thomas Barlow, Dr. Gee, Dr. C. Theodore Williams, and Mr. Alfred Willett, was appointed to settle, in consultation with the Solicitor to the Society, the form of the Debenture and Trust Deed.

This Committee decided that not more than £30,000 was to be thus raised.

The Building Committee appointed at the Special General Meeting on March 11th, 1889, immediately commenced their work, and Mr. Holmes was appointed Chairman.

Messrs. Lake, Beaumont, and Lake were requested to act as Solicitors to the Society, and Mr. William Flockhart as Architect.

Mr. Flockhart was requested to prepare plans and obtain estimates for the required alterations and additions.

The Society then petitioned the Corporation of the City of London for a more secure and advantageous tenure of that part of the property held on lease from the Corporation, and on which it was desired to build; and Deputations, consisting of the President and other Fellows of the Society, attended in support of that petition. The matter having been referred to the City Lands Committee, the Corporation decided, in consideration of the scientific character of the Society, to grant a lease of eighty years at the annual rent of £30; and the Council desire to ex-

press their grateful appreciation of the consideration and liberality of the Corporation.

On April 17th the Committee accepted an offer of £450 a year for the remainder of the Society's lease of the premises in Berners Street, from Messrs. Phipps and Dawson, electrical engineers, possession to be given on September 29th, 1889.

The purchase of the property in Hanover Square was completed on May 10th.

The decision of the Corporation as to the Leasehold portion was received on June 13th.

The builders' estimates for the alterations were received on June 25th, and that of Mr. Nightingale for £6840 was accepted. The contract for the work according to the plan and specifications of Mr. Flockhart was signed on July 9th, and the work was commenced forthwith.

It will be seen, therefore, that no time was lost in preparing the new premises for the use of the Society, and the builder's contract provided for the completion of the work by October 7th.

Unfortunately various events which it was impossible to foresee interfered with the speedy completion of the work which had been hoped for. Prolonged and unavoidable delay was caused, soon after the work was begun, by the Dock Strike, which interfered with the delivery of important iron-work without which it was impossible to proceed. Subsequently legal difficulties were raised by the occupant of the adjoining premises, with regard to the new North Room, and the arrangement of this matter caused some delay.

Moreover, as the work progressed certain alterations in the original plan became inevitable.

The Council greatly regret the inconvenience which the Fellows must necessarily have suffered from the delay in the completion of the premises, and especially from the prolonged closure of the Library, which the most unremitting efforts on the part of the Building Committee were unable to prevent.

But the Council also believe that the Fellows of the Society will appreciate the many difficulties to be overcome, in adapting, altering, and adding to a large house, in accordance with the somewhat complicated requirements of the Society and its tenants, and that it will be felt that no labour or care has been spared by the Building Committee to bring the work to a satisfactory completion in the shortest possible time.

The Council are glad to be able to announce that the Societies which had been accustomed to meet in the rooms of the Royal Medical and Chirurgical Society at Berners Street have all become tenants in Hanover Square, and that numerous additional Societies have also been accommodated.

Arrangements have also been made for building, on the site of the stables, premises which will be rented by Messrs. Webb Miles & Co.

The following is a list of the tenants which the Society has provided for:

The Pathological Society,
The Clinical Society,
The Royal Microscopical Society,
The Gynæcological Society,
The Obstetrical Society,
The Quekett Microscopical Club,
The Society for Relief of Widows and Orphans of Medical Men,
The British Nurses' Association,
Messrs. Belcher and Pite, architects,
Mr. Edwin Ashdown, music publisher (basement),
Messrs. Webb Miles (site of stables),

producing a yearly rental of £1432 10*s.*; 53, Berners Street being let to Messrs. Phipps and Dawson for the remainder of the lease at an annual rental of £450.

The large hall, as well as the North Room, can be let when desired for meetings without interfering with the ordinary business of the Society.

The annexed plan will show the arrangement of the premises.

The lighting is by electricity, a low-tension (100 volts) current being produced by a Kapp-Allen dynamo, worked by a 6-horse power Robey's steam engine. The electricity is stored in an accumulator of 53 cells. The ground-floor is also supplied with gas.

A fire main and hose is provided on every floor.

The drainage has been completely renewed in accordance with the best sanitary requirements.

The Council wish to express their cordial appreciation of the great courtesy and liberality with which the Medical Society, through its President, Dr. C. Theodore Williams, offered the use of its rooms for the meetings of the Royal Medical and Chirurgical Society, and the Pathological and Clinical Societies during October and November, an offer which the Council would have gratefully accepted had it not been possible to make temporary provision for the meetings in the front Library Room at 20, Hanover Square.

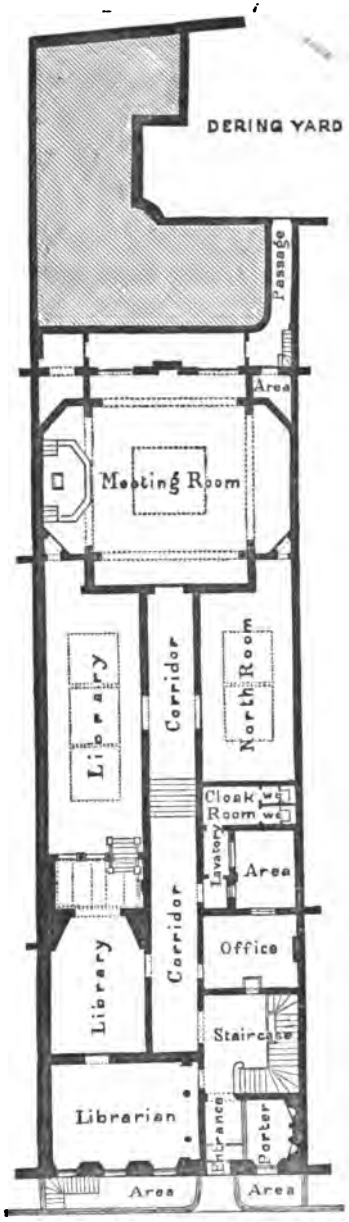
The Council also wish to acknowledge the courtesy with which the Pathological and Clinical Societies deferred their first meetings, and their kind toleration of the temporary arrangements necessarily made for the first few meetings of the Session.

The Council wish to draw attention to the very liberal donation of Dr. Quain to the Society; who, when the necessary funds were being raised for the purchase and alteration of the new house, wrote as follows:

> "I enclose a cheque for £50 as a contribution to the funds of the Royal Medical and Chirurgical Society. My hope is that I shall be thus rendering more useful service to the Society than by becoming a bondholder."

On the receipt of this a resolution was unanimously carried "That the warmest thanks of the Council be given to Dr. Quain for his kind donation to the Royal Medical and Chirurgical Society."

Ground Plan of
The Royal Medical & Chirurgical Society's House
No: 20 Hanover Square



Wm FLOCKHART
Archt.

At a subsequent meeting of the Council it was resolved that Dr. Quain's donation of £50 "be invested as the nucleus of a fund to be called the "Permanent Endowment Fund;" that the interest of that fund be used in such a manner as the Council shall from time to time order, but that the capital of the fund shall under no circumstances whatever be alienated."

This resolution having been communicated to Dr. Quain, he again wrote expressing his gratification that his donation of £50 was to be devoted to the foundation of an Endowment Fund, and offering for the acceptance of the Society a further contribution of £50 to be added to his previous donation.

This the Council most gratefully accepted, and were thus enabled to start the "Permanent Endowment Fund" with the amount of £100.

To this the Council have the pleasure of announcing there has recently been added £50 by Mr. Edward Law Hussey, of Oxford, who has most generously made over to the Endowment Fund the £50 which he had advanced as a bondholder.

It is needless to point out the great advantage to the Society of possessing a fund of this kind, of which only the interest can be used, and to which any one wishing to add to the permanent stability and welfare of the Society can make either gift or bequest.

Its establishment may also perhaps help to dispel the erroneous idea that seems to some extent to have prevailed, that the Society was already possessed of funded capital.

The Council have also the pleasure of acknowledging the valuable gift by Sir Edward H. Sieveking, President, of a jewel consisting of a copy in gold and enamel of the Society's seal surmounted by a crown, to be worn as a badge of office by the President whenever he presides over or represents the Society.

The Council have decided that the retiring President, as his last official act, shall invest his successor with the badge.

Particulars of the income and expenditure of the Society are set forth in the accompanying Statement of Accounts. (p. cxxii).

The number of subscriptions received during the past year was 368, and seven composition fees have been paid.

Forty-three new Fellows have been elected, of whom thirty-two are resident and eleven non-resident. One Fellow has resigned. The Society has lost during the same period seventeen of its Fellows by death.

The total number of the Fellows is at present 793.

The Hon. Librarians report as follows:

Report of the Honorary Librarians, SAMUEL J. GEE, M.D., *and* J. W. HULKE, F.R.S.

"Of the ordinary work of the Library for the year 1889 there is little to record.

"At the very time that last year's Report was presented the movement had begun which, though for the great ultimate advantage of the Library, had the immediate effect of interfering with its work. Trusting that the terms of the Building Contract would be faithfully carried out, and that the new Library Rooms would be ready for occupation at the end of September, we instructed the Resident Librarian to make early arrangements for the packing of the books during the August Recess.

"This work was carried out with great care, and in such a manner that had the new rooms been ready by the time promised, the books could have been placed and ready for use within the space of a fortnight.

"But all our arrangements were rendered valueless when, instead of moving the books to Hanover Square, it became necessary to warehouse them with Messrs. Taylor, of Pimlico. As soon as a single room of the new house was ready no time

was lost in filling it with books and throwing it open to the use of Fellows; but as it was impossible to make a selection of books without reducing the Library to chaos no great advantage was derived from this effort, though the use of the current English journals seems to have been appreciated by many. The period of deprivation is now, however, we trust, at an end, and we hope that by the time this Report reaches the hands of the Fellows all the books will be upon the shelves, and that all the privileges of the Library will be fully available.

"When all the advantages of our magnificent new premises are appreciated by the Fellows, we feel sure the privations of the last few months will seem but a trifling price to pay for so great a boon. For years those actively interested in the work of the Library have looked forward with somewhat gloomy foreboding to the time when the last shelf should be filled; and there seemed no chance that the *res angusta domi* of the Society would ever permit us to pull down our old barns and build greater.

"And now, the reflection that in less than a year we have exchanged increasingly inadequate premises, held on an almost extinguished tenure, for a *freehold* house, capable of accommodating 200,000 volumes, is surely a cause for gratifying encouragement to all those who rightly regard our Library as the most precious possession of the Society.

"The financial aspect of the change will be referred to in the general report, but we may be permitted to congratulate the users of the Library on a fact of hopeful significance in this connection, viz. that the excellent management of the Building Committee has placed the Society in its new house *absolutely rent free.*

"We cannot close this report without placing on record our deep sense of the invaluable services the Resident Librarian, Mr. Mac Alister, has rendered to the Society under very exceptional and most trying circumstances."

SAMUEL J. GEE,
J. W. HULKE,
Hon. Librarians.

By a recent decision of the Council the Library will in future be open from 2 to 7 instead of as heretofore from 1 to 6, which it is hoped will be for the convenience of the Fellows.

During the past session a Scientific Committee has been appointed (in accordance with Bye-Laws, Chap. XV) for the purpose of investigating questions of importance in reference to the climatology and balneology of Great Britain and Ireland.

The following is a list of the members of this Committee:—Dr. Ord (Chairman), Dr. A. E. Garrod (Secretary), Dr. Ballard, Dr. Mitchell Bruce, Dr. Cheadle, Dr. Dickinson, Dr. W. Ewart, Dr. Maguire, Dr. Norman Moore, Dr. Murrell, Dr. Penrose, Dr. Fredk. Roberts, Dr. Fredk. Taylor, Dr. Symes Thompson, Dr. Hermann Weber, Dr. Theodore Williams, Mr. Malcolm Morris, Mr. Fredk. Treves.

The Council have had under their consideration the mode of dealing with papers submitted to the Society for reading, in consequence of the following letter received from Dr. R. J. Lee:—

"6, SAVILE ROW, W.;
March 30th, 1889.

"To WARRINGTON HAWARD, Esq.,
Hon. Sec. of the Roy. Med. Chir. Soc.

"DEAR MR. SECRETARY,—I venture to draw the attention of the President and Council of your Society

to the subject of the standing orders which refer to the *reading* of papers presented to the Society. On the presentation of a paper to the Society it is provided by No. II, Chap. X, p. 8, Standing Orders that 'the Secretaries shall present all papers received by them to the next ensuing Council, but may, if they deem it expedient, previously submit them,' &c.

"According to the Bye-Law of the Society, p. 24 (Chap. XIII, Sec. VI), it is quite clear that the referees are appointed by the Council 'to report to them confidentially on the merits of papers READ before them.' There is no distinct provision made here in regard to papers *not yet read*, beyond the voluntary action of the Secretary implied in the words 'may, if they deem it expedient, previously submit them,' &c. This power given to the Secretary requires consideration, as it leaves the Secretary open to possible suspicion, in regard to the *reading* of a paper, unfair to him and prejudicial to the interests of the Society. On the presentation of a paper to the Society—

"1. Acknowledgment of its receipt ought to be made by the Secretary.

"2. The paper should be laid before the Council before being submitted to any referee.

"3. Referees ought to be named by the Council to report upon whether the paper is likely to contribute to the purposes for which the Society was instituted, as defined by Section I (Bye-Law XI, Chap. I).

"4. No alteration should be made in paper *previous to reading*, providing that the paper contains no objectionable matter and is not too long—that is, beyond the length agreed upon by the Council. It does not seem desirable that referees on a paper should have the power to suggest any alterations in the form of a paper. It is proper that the

author should submit to the criticism which he will receive when the paper is read, and that no criticism should be allowed by referees previous to the reading of the paper.

"I leave this matter in your hands as agreed upon in our interview previous to the last Annual Meeting.

"I am, dear Mr. Secretary,

Ever truly yours,

ROBERT J. LEE."

A Committee was appointed to report upon the matters referred to in this letter, and this Committee was subsequently added to, and the scope of its inquiries enlarged so as to include other questions connected with the selection of papers for reading and publication.

As the result of the reports of the Committees and of the careful consideration of the matter by the Council, it was resolved to recommend to the Annual Meeting the following addition to the Bye-Law No. VI, Chap. XIII, page 24:—"And in special cases as to whether papers forwarded to the Society are suitable for reading before the Society," which will then include in the duties of the referees the consideration of papers at the discretion of the Secretary or Secretaries before they have been presented to the Council for acceptance.

The Council did not think it desirable to make any other changes in relation to this matter.

Finally, the Council desire to express their appreciation of the great services rendered to the Society by those gentlemen who have undertaken the arduous duties of the Building Committee, and especially to Mr. Holmes, the Chairman of that Committee; and in this respect also to record in the most emphatic manner the unfailing energy, the incessant care, and the unremitting devotion to the interests of the Society displayed by the Resident Librarian, Mr. J. Y. W. Mac Alister, to whom, as already stated, it is greatly due that the Society has obtained its present residence.

Mr. Bostock (Treasurer) wished to explain that the account presented by his colleague and himself was necessarily deficient, inasmuch as it gave no statement of the expenditure upon the building. It had been thought to be only misleading to give a tentative statement on this subject, and that it would be better to reserve the whole until the building operations were completed and an accurate and final account could be prepared. The amount expended was somewhat in excess of that anticipated, but, on the other hand, the income secured by such expenditure was about double of the amount originally estimated.

Dr. Hare (Treasurer) observed that the Fellows might be surprised at noticing the abnormally large balance which remained to credit of the general account, but he trusted they would not run away with the idea that this was a genuine surplus; for though it was true that owing to the Library having been closed for nearly a year the expenses under that head had been curtailed, there had been expenses on other matters which quite swallowed up any such saving. The simple explanation of the balance was that a considerable amount of bills remained unpaid at the end of the year when the balance was struck, chiefly owing to the difficulty of getting them in and dealing with them during the exceptional pressure of unusual work upon the officials. With regard to the Building Account, as was always the case with building accounts, the expenditure had exceeded the estimate; but he was happy to be able to assure the Fellows that in this case every penny expended brought them a most substantial and profitable return. The Committee could have completed its work without expending a penny beyond that originally estimated, and have done all that was asked of them; but more than once they had before them the inducement of obtaining such an excellent return for a slight excess on the sum originally estimated, that in the interests of the Society it would have been penny wisdom and pound foolishness to have hesitated. Mr. Mac Alister had proved an excellent agent in securing profitable tenants; for example, it had actually been decided

to let the leasehold stables at the back for £120 when Mr. Mac Alister secured a tenant who, after some negotiation, agreed to pay the Society £350 a year if they would build him new premises on the same site at a cost of some £1300—that is to say, a net increase of £230 was secured on a lease of sixty-seven years for an expenditure of £1300. Surely there could be no question as to the wisdom of accepting such an offer, though it meant exceeding the amount of the Debenture Fund. As they were limited to £30,000 for this fund, it would probably be necessary to find the balance elsewhere; but there would be no difficulty on that score. Dr. Hare also referred to the Permanent Endowment Fund which Dr. Quain's gift had enabled the Council to establish, and which had since been added to by the gift of his debenture by Mr. Hussey, of Oxford, and he urged that the example of the last-named gentleman was an admirable one which might be commended to the attention of other debenture holders, who would thus save themselves the anxiety and trouble of looking out every half-year for the interest cheque of his colleague and himself.

Sir WILLIAM SAVORY said he had great pleasure in moving "That the Report of the President and Council, together with the Treasurers' audited statement of accounts, be adopted and published in the next volume of the 'Transactions.'" He was sure those present heartily congratulated the Council upon thus meeting in their new home.

Dr. STEPHEN MACKENZIE seconded the resolution, which was carried unanimously.

The PRESIDENT moved "That the very hearty thanks of the Royal Medical and Chirurgical Society be given to the members of the Building Committee for their arduous services and the unremitting care which they had devoted to the interest of the Society; services which had resulted in placing the Society in possession of magnificent premises in every way adapted to its requirements and upon the most advantageous terms."

Mr. Hulke expressed the pleasure he had in seconding the resolution. He remarked that the premises in which the Fellows were now assembled formed the best testimony to the Council's work during the past year, and nothing he said could improve upon the eloquence of that fact.

The resolution was carried by acclamation.

Dr. Julius Pollock said he had been asked to move the following resolution, which had already been mentioned in the Report of the Council, viz.: "That the following addition be made to the Bye-Laws, Chapter XIII, sect. 6:—'And in special cases as to whether papers forwarded to the Society are suitable for reading before the Society.'" This might not seem much to have come out of several meetings of a sub-committee, and the careful consideration of certain matters very properly brought before the notice of the Council by Dr. Robert Lee; but it was hoped that the proposed alteration would meet the views of the Society, and there did not appear to be any reason or advantage in making further changes.

Dr. R. J. Lee, in seconding the resolution, said it was important for the Fellows to understand distinctly the object of the change in the Bye-Laws proposed by the Council. It was to give certain powers to the Secretaries and the Referees which they did not at present possess. The Referees were appointed to decide whether papers which had already been read should be printed in the 'Transactions' or not; but they had nothing to do with papers which had not been read. It was now proposed that before a paper was read it should be within the power of the Secretaries to submit it to the Referees, to decide whether it should be read or not. It remained with the Fellows to decide this question as they considered best for the interest of the Society, and he was sure that before making the proposal the Council had given the matter their long and serious consideration.

The resolution was then put to the meeting and carried.

The PRESIDENT then delivered his Annual Address.[1]

Dr. QUAIN said that the privilege had been given him of proposing a vote of thanks to the President for the address to which they had just listened. The address testified to the care and the ability with which the biographical details in the lives of deceased Fellows had been compiled, whilst those present recognised the feeling and the eloquence with which these details were brought before them. Dr. Quain congratulated the President on having held office during this great event in the history of the Society, namely its movement into its present grand abode. He had the greatest possible pleasure in moving "That the hearty thanks of the Society be given to the retiring President, Sir Edward Sieveking, M.D., LL.D., for his conduct in the Chair during his term of office, for his zealous and valuable services to the Society, as for the Annual Address now delivered."

Mr. GEORGE POLLOCK stated that he heartily echoed Dr. Quain's congratulations. During his own term of office in the Chair he had himself made some enquiry with a view to the Society purchasing the house in which they were now established, but at that time there were difficulties in the way which seemed insurmountable. He was delighted that a way had been opened so soon. He had special pleasure in seconding Dr. Quain's resolution.

Dr. QUAIN then put the resolution to the meeting, and it was carried by acclamation.

On the motion of Dr. CHEADLE, seconded by Mr. LANGTON, it was carried "That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Octavius Sturges, Mr. Morrant Baker, and Mr. Christopher Heath for their services to the Society during the past year."

On the motion of Mr. MACNAMARA seconded by Dr. BUZZARD, the following resolution was carried unanimously: —"That the best thanks of the Society be given to the retiring members of Council, Dr. Lauder Brunton, Dr.

[1] See p. 1 of this volume.

Hughlings Jackson, Dr. Ralfe, Mr. Howard Marsh, Mr. Henry Morris, Mr. R. W. Parker, Mr. Edward Tegart, and Mr. W. J. Walsham for their valuable services to the Society during their respective terms of office."

The PRESIDENT then declared that the ballot showed the following gentlemen as duly elected Officers and Council for the ensuing year :—

President.—Timothy Holmes.

Vice-Presidents.—Robert Barnes, M.D. ; J. Langdon Down, M D. ; Alfred Willett ; John Croft.

Treasurers.—Charles John Hare, M.D. ; John Ashton Bostock, C.B.

Honorary Secretaries.—Frederick Taylor, M.D. ; J. Warrington Haward.

Honorary Librarians.—Samuel Jones Gee, M.D. ; John Whitaker Hulke, F.R.S.

Other Members of Council.—Walter Butler Cheadle, M.D. ; William Miller Ord, M.D. ; Arthur Julius Pollock, M.D. ; George Vivian Poore, M.D. ; T. Gilbart Smith, M.D. ; William Harrison Cripps ; Clinton Thomas Dent ; Henry Greenway Howse ; Henry Walter Kiallmark ; Herbert William Page.

He then called the President elect, Mr. Timothy Holmes, to the platform, and addressed him as follows :

" Mr. Timothy Holmes, permit me in the first instance to congratulate you on your election to the Presidency of this Society ; and in the second to obey the directions of the Council by investing you with the badge which it is intended that, in the future, the President of the Royal Medical and Chirurgical Society should wear at all meetings of the Society, and whenever on public occasions he represents our commonwealth. It is my fervent wish that you and your successors, when you quit office, may always leave the Society more flourishing than it was when the insignia of Presidency were conferred upon you."

The President then invested Mr. Holmes with the badge,

and presented to him a silver master-key for the use of himself and the future Presidents of the Society, the gift of the Resident Librarian.

Mr. TIMOTHY HOLMES, in reply, said he could find no adequate words in which to express his feelings of gratitude for the unexpected and undeserved honour which they had done him by electing him to be their President. He had thought that his professional life was over, and all idea of professional distinction had passed away from his mind when the Council had unexpectedly asked him to occupy the proud position to which he had just been elected by the Society. Though conscious of his own demerits, he could not refuse, but it was not until that moment that he felt to its full extent the weight of the burden he had taken upon himself. It was at no ordinary period in the history of the Society that he assumed office, a period commencing in that room on that evening. They were embarking upon a new career, and, honorable, useful, and successful as their course had been in the past, it was impossible to avoid feeling that greater things would be expected of them in the future. He felt almost afraid when he reflected upon the additional burden that the Council had laid upon the Society, a burden much heavier than they had ever borne before. For his own part, he would endeavour to promote the interests of the Society to the utmost of his powers, and he should rely upon the assistance of the Council and of the Fellows in that respect. He insisted upon the importance of doing their utmost to increase the number of their Fellows by every legitimate means. Their position was a great and a strong one, and one in which he had every possible confidence, but at the same time it was not as yet altogether a secure one. They had gone to a very great expense in building, for which he would not apologise, for he was sure that the expenditure had been a most useful one in every way. It had, however, left them with a heavy pecuniary burden, and each Fellow ought to labour conscientiously to enable the Society to support this responsibility, especially by obtain-

ing new Fellows. The hour was so late that he would not detain them more than to assure the Fellows again that his best services would be devoted to the interests of the Society. He would wear with pride the beautiful emblem with which their retiring President had invested him, until he should be called upon to hand it over to a more worthy successor.

The meeting then terminated.

DR.	£ s. d.	£ s. d.	£ s. d.
To Balance in hand on January 1st, 1889 :			
Cash in hand	24 17 2		
	21 11 3		
		46 8 5	
,, at Bankers		26 13 10	
,, on Deposit do.		300 0 0	
			373 2 3
,, *Subscriptions, Fees, &c. :*			
326 Annual Subscriptions at £3 3*s.*		1026 18 0	
41 ,, ,, ...		129 3 0	
32 Admission Fees at £6 6*s.*		201 12 0	
4 Composition Fees for 'Transactions' (1 at £6 6*s.*, 3 at £8 8*s.*)		31 10 0	
3 Life Composition Fees		45 3 0	
Fines		0 3 0	
			1434 9 0
,, *'Transactions,' &c. :*			
Sold by Messrs. Longmans		43 14 7	
,, Society		0 12 6	
Catalogue		0 7 6	
'Proceedings'		2 1 10	
			46 16 5
,, *Rents :*			
Pathological Society		78 15 0	
Clinical Society		78 15 0	
Obstetrical Society		29 8 0	
Society for Relief of Widows and Orphans		30 0 0	
Stables		37 10 0	
			254 8 0
,, *Interest :*			
On Consols		90 5 10	
,, Deposit (Bank)		6 3 0	
			96 8 10
			2205 4 6
Less Subscriptions, &c., for 1890, paid in advance in 1889, and included in Subscriptions for 1890			24 17 2
			2180 7 4
Due to Librarian on account of Petty Cash			31 10 11
			£2211 18 3

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

PERMANENT

DR.	£ s. d.
To Dr. Quain's Donation	100 0 0
,, Mr. Hussey's Donation	50 0 0
	£150 0 0

MARSHALL

DR.	£ s. d.
To amount of Stock, December 31st, 1888	599 7 0
,, Dividends for 1889	16 19 5
	£616 6 5

	Cr.	£ s. d.	£ s. d.
By *House Rent, Taxes, &c.:*			
Ground Rent (less tax)		13 13 0	
Imperial Taxes		12 13 0	
Parish Rates		18 7 ½	
Water Rate		4 0 7	
Insurance		7 6 0	
			55 19 11
„ *Lighting and Heating:*			
Gas, Coal, and Chandler		..	40 10 11
„ *Repairs, Furniture, &c.*		..	72 4 4
„ *Meeting Expenses:*			
Refreshments, Waiters, Microscopes, and Lamps		..	18 19 2
„ *Stationery, Postage, &c.*		..	38 8 10
„ *Salaries and Wages*		..	527 0 9
„ *Library:*			
Books, Binding, and Parcels		..	40 2 0
„ *'Transactions' and 'Proceedings'*		..	102 15 6
„ *Sundry Petty Cash Disbursements*		..	9 8 10
„ *Bank Charges*		..	1 4 10
„ *Cash:*			
In hand		33 3 8	
At Bankers		971 19 6	
„ on Deposit		300 0 0	
			1305 3 2
			£2211 18 3

Audited and found to be correct, 11th February, 1890.

OCTAVIUS STURGES, ROBERT WM. PARKER. | JOHN H. MORGAN. H. MONTAGUE MURRAY. FREDERICK TAYLOR, *Hon. Sec.*

ENDOWMENT FUND.

	Cr.	£ s. d.
By Purchase of New South Wales Four per Cent. Stock		100 0 0
„ Balance in Treasurers' hands		50 0 0
		£150 0 0

HALL FUND.

	Cr.	£ s. d.
By Costs of purchasing Stock (£16 17*s.* 2*d.*)		0 2 3
„ Stock, 31st December, 1889		616 4 2
		£616 6 5

ADDRESS

OF

SIR EDWARD H. SIEVEKING, M.D.,

LL.D., F.R.C.P.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1ST, 1890.

FELLOWS OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY!

When we last assembled on the 1st of March, I adverted to the change which was then only looming before us, but which your energy, and the admirable management of the Building Committee appointed by you at the suggestion of the Council on the 4th of March of last year, has made a reality. I trust that you all agree with me that in selecting this new home we have no reason for regret, but that, on the contrary, the larger habitation and the greater convenience of our library and assembly-room, will be a permanent stimulus to all Fellows to more careful study, to more efficient work, and a means of the cultivation of union and strength in our profession. But whatever our trust and confidence in the future of our Society, our first duty now is to recall to our sorrowing memory the work and achievements of those Fellows who have passed away from us to their eternal home since the last Anniversary Meeting.

The first death that I have to record is that of *William*

Henry Octavius Sankey, M.D.Lond., F.R.C.P., who at the age of seventy-five breathed his last at Boreatton Park, Baschurch, Salop, on March 8th last. He became a Fellow of the Royal Medical and Chirurgical Society in 1847, but although a man of eminence, owing to his residence out of town never filled any office amongst us. Dr. Sankey was the son of a medical man who practised at Wingham, in Kent. He studied medicine at St. Bartholomew's Hospital, and after practising for a time at Margate became resident medical officer at the London Fever Hospital. He here worked much with Sir William Jenner, whom he assisted in his important researches, which are so well known, in regard to the differentiation of the various forms of continued fever. By his special investigations into cerebral pathology at this hospital Dr. Sankey laid the foundation for his subsequent reputation as a specialist in the treatment of insanity. A valuable paper in the January number of the 'Medico-Chirurgical Review' of 1853, on the specific gravity of the brain, was the result of researches which he carried on with great care for seven consecutive years. I am happy to be able to quote Sir William Jenner's opinion, that Dr. Sankey was a man of considerable mental vigour and thoroughly honest in searching after truth, well formed in his profession, kind of heart and most estimable.

In 1854 Dr. Sankey left the Fever Hospital and took charge of the female side of the Middlesex County Lunatic Asylum at Hanwell, where he was the intimate and staunch disciple of Connolly, with whom he co-operated to the utmost to establish the humane treatment of the insane advocated by that distinguished physician. In a controversy[1] raised at that time, Connolly, in consequence of a paper written by Dr. Sankey pointing out that harsh words were as inadmissible in the treatment of lunatics as corporal punishment, said to him in a letter, "You indeed really understand what non-restraint means; there are very few that do." Dr. Sankey

[1] Communications from Dr. H. R. O. Sankey, the son of the subject of this notice.

was never a robust man, and the work required of him at Hanwell so told upon him that after a period of ten years he resigned, and practised privately at Landywell Park Lunatic Asylum, in Gloucestershire. Shortly after leaving Hanwell he was appointed Lecturer on Mental Diseases at University College, London, an office that he held for many years. In 1882 he quitted Landywell Park, and removed all his patients to Boreatton Park, Shrewsbury. In 1884 he published the second and very much enlarged edition of his lectures, which first appeared in 1866 under the title of Lectures on Mental Diseases, a work that is still a high authority on the subject it deals with, and during his life made many contributions to medical journals both in England and France.

For several past years Dr. Sankey suffered from some obscure and very painful hepatic disorder, probably biliary calculi. But of late his health had somewhat improved. His death was due to an attack of pneumonia of a few days' duration. His mantle has descended upon his son Dr. H. R. O. Sankey,[2] who is Superintendent of the Connolly Asylum at Hatton, Warwick.

The second obituary notice that I have to submit to you is that of a man who, both in this Society and in the world of medical science, has occupied a very prominent position, *Charles James Blasius Williams, M.D.Edin., F.R.S.*, who at the advanced age of eighty-four ended his useful life on March 24th, 1889, at the Villa du Rocher, Cannes. Dr. Williams was elected a Fellow of the Royal Medical and Chirurgical Society in 1840, held the offices of Councillor in 1849 and 1850, of Vice-President in 1860–1, and of President in 1873–4; he also served as Referee in 1843–4; he was Chairman of the Scientific Committee on Suspended Animation in 1862, and he communicated one paper to the Transactions.[1] It is impossible in the

[1] Title of Dr. C. J. B. Williams' paper in the Transactions, vol. lvii, 1874: "On the Acoustic Principles and Construction of Stethoscopes and Ear-trumpets."

[2] Dr. H. R. O. Sankey has now taken his father's place at Boreatton.

brief space at my disposal to do full justice to the work and influence of Dr. Williams; the former has certainly made an epoch in British Medicine, and will ever constitute an important landmark in the enormous strides of the present century. Dr. Williams, as we gather from his own record,[1] was the youngest but one of nine children of the Rev. David Williams, for forty years perpetual curate of the Collegiate Church of Heytesbury, in Wiltshire. In 1820 the subject of this memoir went to Edinburgh, where he was specially attracted by Professor Hope, the Lecturer on Chemistry, and by Dr. Alison, the Professor of Medicine. In 1824 he took his degree, presenting as his thesis[2] De sanguine ejusque mutationibus, in which he gives a summary of the most recent researches on the properties of the blood and its composition, with the results of his own experiments. In 1825 Dr. Williams went to Paris, where he remained for fourteen months, attending the practice and teaching of Majendie, Dupuytren, Thénard, and others, but especially that of Laennec, to whom the world is mainly indebted for having taken up and developed Auenbrugger's discovery of the value and uses of auscultation. It is interesting to note that Laennec in general maintained the sufficiency of a simple cylinder of wood for a stethoscope, perforated or hollowed out at the pectoral end (whether conically or parabolically did not matter), and fitted with a stopper to be used for certain purposes. As a guide to a better understanding of the works of Laennec, Williams in 1828 published A Rational Explanation of the Physical Signs of Diseases of the Chest. After a temporary residence in the country Dr. Williams in 1827 settled in London, where he enjoyed the friendship of Sir James Clark and Dr. (afterwards Sir) John Forbes. After two trips in charge of invalids, to Madeira

[1] Memorials of Life and Work. By C. J. B. Williams, M.D., F.R.S., 1884.

[2] A copy of the thesis is in our library. It may be stated that his first publication, On the Low Combustion of a Candle, Visible in the Dark, appeared in the Annals of Philosophy, July, 1823.

and to Switzerland, he took a house in Half-moon Street, married, and became a Licentiate of the Royal College of Physicians; of this body he was elected a Fellow in 1840, gave the Goulstonian and Lumleian lectures, and held the offices of Censor and Councillor. Soon after settling in London Dr. Williams wrote several articles for the Cyclopædia of Practical Medicine of Forbes, Tweedie, and Connolly, dealing chiefly with the organs of respiration.[1] In 1833 a second edition of his Diseases of the Chest appeared. It was at this time that the profession were much exercised by the discussion as to the causes of the sounds of the heart, Williams maintaining that the first sound was due to muscular contraction; Hope, on the other hand, attributing this sound to collision of the particles of the blood in the ventricles. In 1835 a third edition of this work issued from the press, and in this year the author was elected a Fellow of the Royal Society. For two years Dr. Williams lectured at the Kinnerton Street School of Medicine, and in 1839 he was appointed successor to Dr. Elliotson (who had created great animosity by the enthusiasm with which he took up the subject of Animal Magnetism) as Professor of the Principles and Practice of Medicine, as Professor of Clinical Medicine at University College, and as Physician to University College Hospital. The fourth edition of his work on the chest appeared in 1840, and in his Goulstonian Lectures at the College of Physicians in 1841 he dealt with topics in general pathology, which he afterwards embraced in his important work entitled Principles of Medicine; of this the British and Foreign Medical Review said at the time: We hail its appearance not only on account of the value we are ready to attach to any production of its accomplished author, but also as the indication of a vast improvement in medical teaching, which must operate most favorably at no distant date upon medical practice, besides giving a stimulus

[1] The chief articles were on Bronchitis, Catarrh, Coryza, Expectoration, Irritation and Counter-irritation, Malformations of the Heart, Obesity, Pneumonia, and the Stethoscope.

to many active and intelligent minds to follow out the inquiry which it has so successfully opened. Those whose studies date back to this period will, I believe, be ready to endorse this opinion. The year 1841 was also marked by the opening of the Hospital for Diseases of the Chest, in Brompton, the foundation of which Dr. Williams had energetically assisted, and of which he was elected Consulting Physician, an appointment which he held to the end of his life. Events that it is unnecessary to dwell upon here led to his resigning the Professorship of University College in 1849, and in 1851 he removed from Holles Street, where he had resided since 1839, to Upper Brook Street, where he remained until he withdrew from practice and from *la brumeuse Angleterre*, to seek renewal of life on the sunny shore of the Mediterranean.

The day after Dr. Williams ended his career, our distinguished Honorary Fellow, *Franz Cornelius Donders*, died, on the 25th March, 1889, at Utrecht, in Holland, accepted not only in his own country, but wherever his works penetrated, as one of the greatest physiologists and scientific ophthalmologists of the age. He was born on May 27th, 1818, at Tilburg, in the south of Holland, the youngest child and only son of his parents. Up to his seventh year his mother was his instructress. The school education which followed appears to have been very inefficient, especially in regard to mathematics. In 1835 Donders commenced his medical curriculum at the University of Utrecht, and at the Military Medical School of the same town. Five years later he was appointed Lecturer on Anatomy and Physiology at the latter institution, and after the lapse of another lustrum he became one of the Professors of the Medical Faculty of the University, Schroeder v. d. Kolk still retaining the chair of Anatomy and Physiology. Donders now established a physiological laboratory, where he taught General Physiology, from which he successively advanced to General Pathology, Forensic Medicine, and Ophthalmology. The last subject forced him, *nolens volens*, into medical practice. The

International Exhibition of 1851 caused him to visit London, where he made the acquaintance and secured the permanent friendship of his great *confrères*, Bowman and von Gräfe. In 1858 Professor Donders founded an ophthalmic hospital.[1] In 1863, after the decease of Schroeder v. d. Kolk, he took entire charge of the physiological teaching of the University, and a large proportion of his predecessor's other work. In 1866 the University established a physiological laboratory, which was opened in 1867, and of which Professor Donders remained Director till 1888. His countrymen, who called him "groot en goed" (great and good), particularly appreciated the fact that, although he received numerous invitations to transfer his services to other universities, he remained faithful to the last to his Alma Mater, the University of Utrecht. Donders, as his biographer Dr. Landolt informs us, possessed in a high degree all the qualities which constitute a perfect teacher; learning as profound as it was extensive, an excellent memory, a capacity of placing himself in perfect sympathy with his audience, a power of making abstract questions intelligible, facility of expression, a sonorous and flexible voice, and an expressive and dignified delivery.

Donders' first great professional merit consisted, not, as has been said, in discovering astigmatism, which must be attributed to Helmholtz, but in rendering Helmholtz's discoveries applicable to practice. The New Sydenham Society deserves the credit of having introduced Donders first to the Medical Profession of England by publishing in 1864 his work On the Anomalies of the Refraction and Accommodation of the Eye. Donders was early in life an adherent and expositor of the then scarcely recognised doctrine of the conservation of force. In 1845 he published an essay on The Exchange of Material as a Source of Heat in Plants and Animals. This was followed in 1848 by an inaugural dissertation on The Harmony of Animal Life, in which he anticipates some of the doc-

[1] Nederlandsch Gasthuis voor Ooglijders.

trines more fully and completely elaborated by Darwin. His micro-chemical researches, published conjointly with Malden (1844-7), are the first of their kind, and were followed by numerous other works of greater or less extent, which have all served to establish Donders'[1] claim to be regarded as one of the most scientific and at the same time practical men of his day. The following remarks, with which Dr. Brailey has favoured me, are an echo of the reputation which Donders enjoys in Great Britain:

In appreciating the scientific labours of Donders it is necessary to bear in mind that, starting as a pure physiologist, he was led to transfer very largely his energies to physiology in its bearing on ophthalmic practice—a field less widely known, and therefore less appreciated. Physiology as applied to the phenomena of vision was indeed in its infancy. Even myopia was most imperfectly understood; knowledge of hypermetropia was absolutely wanting; and astigmatism, though known through the labours of our own Thomas Young and Airy, was absolutely unappreciated in its relation to curative medicine. But not only was little understood of the refraction of the eye, but even its movements were very imperfectly comprehended, and the entire mechanism of accommodation also was involved in mystery. The prolonged labours of Donders showed the alteration which the vertical meridian undergoes in different movements of the eye and head, and defined the effect of individual muscles. Donders was the first to explain the relation of refractive errors to concomitant squint, a subject of enormous and daily increasing importance in relation to the cure of this condition. One of the practical results of his labours was his suggestion regarding tests of colour for railway and marine services at the International Congress at Amsterdam in 1879, of which Donders was President. His investigations on the histology of elastic tissues, and on the rapidity of transmission of nervous impulses, testify to work which alone would

[1] I refer those who desire a fuller account of Professor Donders' life and works to Warlomont's Annales d'Oculistique, tome cii (14e Série, t. ii).

have raised him to a high place in the roll of science. The fact of his being elected a foreign Fellow of the Royal Society, and one of the four honorary members of the Ophthalmological Society, shows that this country did not fail to appreciate him; while the universal esteem which he enjoyed was demonstrated by the celebration which was held on the occasion of his retirement from the Professorship of Physiology in 1888, when men of science from all parts of the civilised world assembled at Utrecht to do him honour. A sum of over £3000 was presented to Donders (£300 of which came from here) on this occasion, which he devoted to the foundation of a travelling fellowship, to be awarded at intervals of eight years to promising students of ophthalmology and physiology.

The next loss sustained by the Society was that of a most accomplished and genial Fellow, personally known to many of you—*Charles Bland Radcliffe*. The scion of an ancient family long settled in the Isle of Man, he was born in 1822 at Brigg, in Lincolnshire. He received his first training from his father, who was a clergyman. Young Radcliffe is stated to have had so much success at the very outset of his studies that at the age of seven years he was able to read Horace in the original. After studying his profession at Leeds, Paris, and London, Dr. Radcliffe took his degree of M.B. at the London University in 1845, and (having obtained the license of the Royal College of Physicians in 1848) the M.D. in 1851, when he married, and was appointed Assistant Physician to Westminster Hospital. With this institution and with the Queen Square Hospital for Paralysis and Epilepsy, to which he was appointed in 1863, Dr. Radcliffe was associated to his end.

The College of Physicians early recognised the merits of Dr. Radcliffe by electing him to the Fellowship in 1858, and appointing him Goulstonian Lecturer in 1860 and subsequently Croonian Lecturer in 1873. He there also held the offices of Councillor and Censor. He joined this Society in 1852, was member of the Council in 1867–8, Vice-

President in 1879 and 1880, Treasurer from 1881 to 1886, and for many years a Referee. Always a great worker, Dr. Radcliffe was indefatigable to the end; he was able on the very last day of his life to see several patients at home in the morning, called for a few minutes at the hospital in Queen Square in the afternoon, and paid a short visit to the British Museum on his way home. He dined quietly, and was engaged in reading when a varicose vein burst; and although the hæmorrhage was arrested, death ensued speedily, probably from shock and failure of the heart. He died at his house in Cavendish Square on the 18th June, 1889.

Dr. Radcliffe's professional career was chiefly marked by his labours and works in connection with diseases of the nervous system. His book on Epileptic and other Convulsive Affections of the Nervous System went through several editions, and he wrote important articles for Reynolds' System of Medicine, on Diseases of the Spinal Cord, on Chorea, and on Locomotor Ataxy. Everywhere he exhibited a thorough knowledge of the subjects he handled, and much originality in his views, which are perhaps nowhere so much shown as in his Vital Motion a Mode of Physical Motion (published in 1876), and in his Behind the Tides, which has only, as yet, been printed for private circulation. In the last essay his object is to prove that there is a tidal wave in the land bearing a definite relation to the tidal wave in the sea, and that the deep-seated subterranean heat also has a definite tidal movement. Dr. Radcliffe was an earnest student of vital dynamics, many of the phenomena of which he solved by reference to electrical force. His fundamental doctrine, as stated in an appreciative estimate by Dr. Burdon Sanderson,[1] was that all the functions or activities of the nervous and muscular systems were essentially electrical. In the fact, he writes, that muscular action is directly proportionate to the development of heat and the exhalation of carbonic acid there is nothing to

[1] 'Brit. Med. Journal,' June, 1889.

justify the notion that heat is transformed into muscular force, or that electricity may not be developed along with heat in the combustion of force-fuel within the system, and that electricity may not do the work that has been ascribed to muscular force. . . . In a word, you may with little or no trouble satisfy yourself that muscular force and nervous influence must share the same fate, and that the only intelligible agent that is left in possession of the field is electricity. Dr. Radcliffe accordingly looked forward to the time when the words irritability, irritation, stimulation, and the like, will be replaced by other words which show that the idea of irritability is resolved into that of natural electricity.

Dr. Radcliffe's creed, writes an old friend of his, embraced medicine, philosophy, and religious thought; but his sympathies were not confined to either of his high subjects. He was interested in the work done by all sorts and conditions of men; and while defending his own views of things, which were at least strikingly original, he was tolerant of the opinions of others. He could appreciate a good novel and delight in a good sermon, and he gained the friendship of many classes of society. The extent and variety of his reading were remarkable, and gave a charm to his conversation that can never be forgotten by those who knew him intimately.

The next Fellow to whose death I have to draw attention was a gentleman of great acquirements, of remarkable independence of character, and one who, but for a singular misfortune, would undoubtedly have long occupied a very prominent position in the profession—*Dr. Thomas King Chambers;* who died after long suffering on August 15th, 1889, at the age of seventy-one. The son of a London police magistrate, and the grandson of Sir Robert Chambers, Chief Justice of Bengal, he received his early education at Rugby under Arnold, and at Shrewsbury under Butler. He graduated in honours as B.A. of Christ Church, Oxford, and took the degree of M.D. at the same University in 1846. His medical curriculum at St. George's enabled Chambers

to publish the first work which attracted the attention of the medical profession, the Decennium Pathologicum, giving an analysis of the hospital post-mortem records for ten years. It appeared in a series of papers in the British and Foreign Medico-Chirurgical Review, a periodical now unfortunately extinct, to which Dr. Chambers before and during my editorship was a frequent and valued contributor. The vigour and sincerity of his style was to me always very refreshing. Having been elected to the Fellowship of the College of Physicians in 1848, he was, at the opening of St. Mary's Hospital in 1851, appointed one of three Senior Physicians, and from the opening of the school shared in the chair of Medicine. At the College of Physicians Chambers held the Goulstonian Lectureship in 1850, the Lumleian in 1863, and delivered the Harveian Oration in 1871. After the death of Dr. Rolleston, Dr. Chambers was in 1881 appointed the representative of Oxford on the Medical Council; but his health had already at that time been undermined, so that he was no longer able to bring to bear on educational questions the energy and clear-sightedness which had long made him a valued adviser on this and allied subjects. Among his experiences should be mentioned his journey with the Prince of Wales in 1859, whom he accompanied as physician through Italy, Spain, and the north of Africa, and who, on the establishment of his household, appointed him his Honorary Physician. The outcome of this expedition was a small book on the Climate of Italy; but Dr. Chambers' chief claims to literary and professional distinction rest upon several works in which he treated of diseases of the stomach, of diet, and regimen. This Society enrolled him as a Fellow in 1844, and he contributed to its Transactions one very interesting paper, in 1854, on Mollities Ossium. He successively filled the offices of Councillor, Vice-President, Librarian; and for many years was a Referee. Much as there was in Dr. Chambers to admire as a physician, as a teacher, as a professional and general writer, as an author and artist (for he was eminent as a draughtsman, painter,

and sculptor), nothing is so touching in his life and character as the heroism with which he bore the disappointment to which he was doomed, and the sufferings that he was called upon to undergo. In this, as in general culture, he may serve us all as a model. In the year 1864, having previously alarmed his friends occasionally by symptoms connected with an enfeebled vascular system, he was found to have a popliteal aneurysm, which necessitated the removal of the left leg. Undaunted by a loss that would have utterly cast down men of a feebler mental constitution, Dr. Chambers continued the active pursuit of his profession, serving as Examiner at Oxford and Durham, attending the Hand-in-Hand Assurance Company, assisting at the Medical Council and at the Medical School for Women in Henrietta Street, Brunswick Square, and working loyally and energetically wherever he could be of use, until, nine months before his death, the carotid arteries both exhibited aneurysms, while at the same time serious cardiac complications declared themselves. From this time to his death, in spite of every care and attention, his life was one prolonged agony, during which, we cannot doubt, his strong religious convictions were a solace, and opened out to him a brighter and more enduring refuge.

Those who were most intimately acquainted with Dr. Chambers traced a distinct resemblance between his character and that of Oliver Cromwell, who was one of his direct ancestors. John of Gaunt, "time-honoured Lancaster," was another man of great power who occurs among the ancestry of our friend. We who knew Dr. Chambers personally feel assured that, like all assiduous workers, he would join with Browning in saying:

> I count that heaven itself is only work
> To a surer issue.

Dr. Chambers in 1847 married the second daughter of Mr. Maitland, of Loughton Hall, Essex, who with two daughters, one of whom is married to Mr. Ouless, the Academician, survives him.

A pupil of Dr. Chambers, and therefore a much younger man, follows him in the funereal list that I have to submit to you. *Walter John Coulson, F.R.C.S.*, died, after a brief illness, on April 30th, 1889, at the early age of fifty-five. He received his medical education at St. Mary's Hospital, where he successfully filled the offices of House Surgeon, Curator to the School, and Assistant Surgeon. He was also attached to the Lock Hospital. Being specially attracted by a branch of surgery in which his uncle, Mr. William Coulson, formerly Senior Surgeon to St. Mary's Hospital, was eminent, he assisted in the foundation of St. Peter's Hospital for Stone, of which at the time of his death Mr. Coulson was Senior Surgeon. Besides editing his uncle's work on Diseases of the Bladder and Prostate, he published, in addition to other surgical papers, a work entitled: Stone in the Bladder; its Prevention, Early Symptoms, and Treatment by Lithotrity, as well as A Treatise on Syphilis. Besides enjoying an excellent reputation as a surgeon and writer, Mr. Coulson's character and amiability secured him many attached friends, who deeply deplored his early demise. Having inherited a a large fortune from his uncle, it is the more to his credit that he was devoted to his work, while it enabled him to enjoy thoroughly the various sports which can only be legitimately indulged in by those whose income does not depend only upon professional sources. His chief characteristics, an intimate friend of his informs me, were his buoyant spirits, his love of outdoor exercises, and his extreme generosity, which he indulged in largely. He had a remarkable influence over his patients, and a large number of them became his warm friends. To this Society Mr. Coulson was elected in 1864.

The next loss sustained by this Society and by the Profession of Medicine was that of *Samuel Osborne Habershon, M.D., F.R.C.P.*, first a distinguished pupil of Guy's, and subsequently one of the most eminent of the physicians of that world-renowned hospital. He was born in 1825 at Rotherham, in Yorkshire, and died on the 22nd August,

1889, of ulceration of the stomach, at the age of sixty-three. Dr. Habershon had the advantage of belonging to an excellent stock. Some of his ancestors emigrated to America, where at least two of them occupied prominent posts in the early days of the North American republic. He himself entered at Guy's Hospital in 1842, and there and at the University of London subsequently, he enjoyed continued success. In the first M.B. examination at the latter institution he secured no less than three gold medals and two exhibitions. His further successes at the second M.B. and at the M.D. examination secured him the Lectureship on Comparative Anatomy at Guy's in 1851; he subsequently became Lecturer on Pathological Anatomy, and in 1851 was appointed Assistant Physician to the hospital. After his teacher Dr. Addison's death, he became Senior Physician in 1873, and Lecturer on Medicine. A painful conflict between the authorities of the hospital and the medical staff regarding the internal administration of the Institution, into the details of which it is unnecessary to enter, caused in 1880 the resignation by Dr. Habershon and Mr. Cooper Forster of their connection with Guy's, a severance which appeared unavoidable at the time, but in which the sympathy and approval of the entire medical profession were with the medical officers. Among the many offices that Dr. Habershon occupied, apart from his hospital, it is specially our duty to remember him here, where, besides giving three important papers to our Transactions,[1] he filled the post of Secretary in 1867, that of Councillor in 1869–70, and of Vice-President in 1881–2, and was Referee almost through the whole period of his connection with the Society, when not holding a post with which this latter office is incompatible. This alone is a clear sign

[1] The titles of Dr. Habershon's papers in our Transactions are—On the Etiology and Treatment of Peritonitis, vol. xliii, 5; Clinical Observations illustrating the Effects produced by the Implication of Branches of the Pneumogastric Nerve in Aneurismal Tumours, vol. xlvii, 35; and Acute Poisoning by Phosphorus, Jaundice, Death on the Fifth Day; Fatty Degeneration of the Liver, &c., vol. l, 87.

of the estimation in which his knowledge, his integrity, and judgment were held.

The College of Physicians showed their recognition of Dr. Habershon's merits by electing him to the Fellowship in 1856. He served the various offices of Examiner, Councillor, and Censor at different times, and was Vice-President of the College in 1887. He delivered the Lumleian Lectures On the Pathology of the Pneumogastric Nerve, at the College of Physicians in 1876, and the Harveian Oration in 1883. Besides numerous contributions to the Guy's Hospital Reports, which all exhibited much careful observation and research, Dr. Habershon attracted the special attention of the medical profession by his various works connected with abdominal disease, among which his Pathological and Practical Observations on Diseases of the Abdomen, and his work On Diseases of the Stomach, are probably the most widely known and appreciated. Not satisfied with the many claims that his professional position made upon him, Dr. Habershon devoted both time and money to the furtherance of charitable work, to which his strong religious convictions especially impelled him. Never robust, he enjoyed fair health until a year and a half before his death, when he was attacked by severe dyspepsia, from which he was recovering when his wife's death in April caused a relapse, and this ended in ulceration of the stomach, hæmorrhage, and death. One son and three daughters survive to deplore the loss of an excellent father, an eminent physician, and a self-sacrificing citizen.

Dr. Charles Elam, F.R.C.P., died on the 20th July, 1889, at the age of sixty-five. Born in Birstall, near Leeds, his father, a Wesleyan minister, supervised his early education. He went through his medical curriculum at the Leeds School of Medicine, and took the degree of M.D. at the London University in 1850, where he distinguished himself in physiology and comparative anatomy, in surgery, in medicine, and in midwifery. After graduation, Dr. Elam served in the Leeds Infirmary as House Surgeon, and

then settled for twenty years at Sheffield, where he lectured on medicine and physiology at the School of Medicine, and was appointed Physician to the Infirmary. In 1868 Dr. Elam migrated to London, where for a short time he was connected with the Hospital for Paralysis in Queen Square. In 1869 Dr. Elam became a Fellow of this Society, and served on the Library Committee in 1886–8. In 1870 he was elected to a Fellowship of the Royal College of Physicians, of which he had become a member in 1860. Throughout his life an ardent student, Dr. Elam was a frequent contributor to the literature of our profession, and his works, though occasionally the cause of controversy, had many admirers. He wrote numerous papers on subjects connected with disorders of the nervous system for the Journal of Psychological Medicine. On Illusions and Hallucinations, A Physician's Problems, On Cerebral and other Diseases of the Brain, The Gospel of Evolution, are some of the more important works from his pen. Judging from what I have read of Dr. Elam's writings, I consider him a man of large and extensive gifts, possessing sound classical knowledge, while capable of appreciating and estimating modern science at its true value. A Physician's Problems would be a valuable addition to the library of all well-educated persons, and I would specially recommend its perusal to every member of the Society for Psychical Research.

His last illness, which commenced in November, 1888, was a long and weary one, born with fortitude and gentleness. It commenced with solid œdema of one leg, due to phlebitis of the deeper veins; the superficial veins becoming involved caused much suffering, and the disease gradually extended to the other leg, and then to the upper extremities. There was little constitutional disturbance throughout, and Dr. Elam retained full possession of his faculties till within a few days of his death.

Dr. Cumberbatch, who was only admitted to the Fellowship of the Royal Medical and Chirurgical Society in the year of his decease, died on the 18th August last, after an

illness of but a few hours' duration, of angina, apparently the result of the overwork which is so frequently the cause of fatality in our ranks. *Laurence Trent Cumberbatch*, born in Barbadoes on May 1st, 1824, studied medicine at Dublin, became M.R.C.S.Eng. in 1848, and after joining a general practitioner at Chipping Norton, came to London, where his ability and tact were speedily recognised, and brought him into an extensive, chiefly obstetric, practice. Sir Charles Locock, having a high opinion of him, put many opportunities in his way. He took the degree of M.D. at St. Andrews in 1866, and in the same year became a M.R.C.P. About seven years ago he found that work was undermining his powers, and, under advice, abandoned a large portion of his practice; but this and the greater relaxation he allowed himself did not suffice to stave off the fatal issue at the comparatively early age of sixty-five. Dr. Cumberbatch was much appreciated in and out of the profession; generally liked on account of his thorough honesty, honorable conduct, unselfish and kindly disposition and demeanour; his sympathetic and successful behaviour to his patients especially endeared him to the denizens of the sick room.

Dr. Cumberbatch leaves a widow, two married daughters, and three sons to mourn his loss.

The 7th November, 1889, was the day on which *Henry Haynes Walton, F.R.C.S.*, breathed his last at his house in Brook Street, where he had resided over thirty years. The youngest son of the Provost-Marshal of Barbadoes, he was born in that island on March 3rd, 1816. His mother, the daughter of General Haynes, was remarkable for talent and force of character; the energy and perseverance which characterised the subject of this brief memoir are supposed to have been especially derived from her. Great reverses in the family compelled him to enter upon a more lucrative profession than that he had been intended for, and he was, after his widowed mother had come to London, entered as a student at St. Bartholomew's Hospital, where his diligence and perseverance soon brought him under

the notice of Sir William Lawrence, who thought and spoke highly of him. After filling the post of House Surgeon at St. Bartholomew's, Walton in 1851 became Asistant Surgeon to the then recently established St. Mary's Hospital; subsequently Lecturer on Anatomy and Operative Surgery at the School, Lecturer on Ophthalmic Surgery, and, after his withdrawal in 1886, Consulting Surgeon to the Hospital. When he accepted the post of Surgeon to the Ophthalmic Department of St. Mary's, Walton gave up his connection with the Central London Ophthalmic Hospital in Calthorpe Street, which he had founded about 1851, and of which he remained Consulting Surgeon to the last. Although he published numerous papers on surgical subjects, and was distinguished as a general surgeon, his special taste, as may be gathered from what has preceded, lay in the direction of the ophthalmic branch of the profession. His chief work, entitled A Practical Treatise on Diseases of the Eye, went through three editions, the first appearing in 1853, the second in 1861, and the last, very much enlarged, in 1875. In an elaborate article on Mr. Walton's first edition, by Dr. Mackenzie, in the Medico-Chirurgical Review of 1853, it is spoken of in the following terms:—In the whole range of ophthalmological literature we know of no work which, on the whole, better deserves a place in the library of the surgeon than the treatise of Mr. Haynes Walton. It is full of sound practical views, and shows the rapid advances which are being made in this department of the medical art. Most of the cases related have occurred to the author himself, and prove him to be an observing and able practitioner. His style is good, being perspicuous and unaffected. A leading professor of ophthalmology of the present day essentially confirms these views of Walton's work, stating what as a physician I should consider high praise, that he regards the diseases of the eye from a general point of view, and not from a special one. In regard to his writings generally it may be said, the same authority concludes, that the descriptions

of disease and the mode of performing operations are concise and clear, whilst the treatment advised is always sound and good. Without being a great, he was an intelligent, thoroughly reliable, and honest ophthalmic surgeon. Of this Society Mr. Walton became a Fellow in 1851, and he gave one paper to the Transactions, entitled Pathological Remarks on the kind of Palpebral Tumour usually called in England Tarsal Tumour.

A distinguished hospital surgeon who was on intimate terms with him for many years speaks of his knowledge of regional anatomy and his skill as an operator with the highest praise. He remarks that Walton enjoyed unusual success in his operations, and that his judgment in diagnosis was remarkable. His advice in railway cases was of great value, and as surgeon to the Brighton and South Coast Railway he is said to have saved the Company many thousands of pounds by the readiness with which he detected fraud and malingering, an item that has so often to be reckoned with after railway accidents. As a clinical teacher his style was short and impressive, and consisted for the most part (as I have been informed by a distinguished surgeon who has attended them) in the giving forth of practical hints which were the offspring of his own wide personal experience. He would hit off the prominent features of an obscure or interesting case, and succeed in putting them before the student in such a way as to make a lasting impression.

Mr. Walton was a man of robust physique and fond of hunting. Twice married, his first wife, the daughter of the Hon. John Reed, of New Court, Gloucestershire, bore him numerous children, four of whom died, at different ages, of diphtheria. Three sons and one daughter survive. Eleven years ago he lost his first wife, and subsequently married Miss Keelan, the daughter of a retired officer of the Naval Medical Department. She also survives him.

On his return from his summer vacation in 1889 Mr. Walton suffered from a feverish indisposition, which he attributed to the insanitary condition of the localities he

had visited. This, however, did not yield to home care and treatment; symptoms developed which unmistakably pointed to the liver being the seat of serious mischief, probably acute yellow atrophy, under which, retaining his mental capacity till within a few days of his death on the 7th November, he sank. He was too ill when we assembled in this building for the first time to attend, but expressed his very warm interest in the event, and his regret at being forced to be absent.

The month of November was also fatal to another of our Honorary Fellows, *Professor Volkmann*, of Halle, in Prussia, one of the most scientific of German surgeons. Richard von Volkmann was born at Leipzig on August 17th, 1830. His father, in 1843, was appointed Professor of Anatomy and Physiology to the University of Halle, and being a man of great general culture, as well as distinguished in his special science, three times filled the office of Rector Magnificus of the University. After enjoying an excellent preliminary education Richard entered the University in 1850, and after taking his degree became assistant to Professor Blasius at Halle. He at once appears to have attracted a large practice, so that his German biographer regards it as marvellous that he could make time for his scientific investigations, his microscopic work, and his extensive and carefully executed professional drawings. His first important monograph, Observations on Certain Tumours that are to be distinguished from Cancer, appeared in 1858. This was followed by papers On the New Formation of Haversian Canals in Osseous Tissue, and in 1865 by his master-work, The Diseases of Bones and Joints. After taking a professional part in the Austro-Prussian war, Volkmann was made Professor of Surgery in the place of Professor Blasius, who retired superannuated. From this time Volkmann's surgical reputation grew from day to day, and his professional work was only interrupted by the Franco-German war, during which he occupied important positions at Mouzon, at Versailles, and at Soisy. He relieved the tedium of the

siege by sending home, under the title: Dreams by French Firesides,[1] poems which have since been published and are much liked in Germany. After the war was over, Volkmann had ample opportunity of examining and carefully testing the antiseptic theory and practice of our great compatriot Joseph Lister. Sceptical at first, he soon became the prominent advocate of Listerism in Germany, of which, at the International Medical Congress in London in 1881, he said, that the new doctrine which has wrought a universal change in surgical treatment, and the new and difficult method which has multiplied the responsibilities of the practitioner extremely, has an assured triumph throughout the civilised world. England, Richard von Volkmann remarks in the address he delivered on that occasion—England may feel proud that it was one of her sons whose name is inseparably associated with the greatest advance ever made by surgery. All other nations may without jealousy award him the crown. For the long, noiseless work which made the ripening of the seed possible, and which we are now harvesting in rich abundance, has been of an international character, and both France and Germany have contributed their share. No one has more liberally acknowledged the value of antecedent workers than Joseph Lister.[2]

Indefatigable in his profession, there was scarcely a year, from the date of his diploma thesis, De pulmonum gangræna, in 1854, to that of his death on November 28th, 1889, that he did not produce some important contribution to surgical literature. His last effort was an address delivered on October 31st of last year On the resection of the Ribs in certain cases of Scoliosis, at the Society of Surgery in Berlin. Volkmann is described as having been a very handsome man, of imposing mien and engaging manners; and though devoted to his profession,

[1] Träumereien an französischen Kaminen, under the pseudonym Richard Leander.

[2] See Report of the Fifth General Meeting of the International Congress in London, in 1881.

able in his vacations and on his travels to devote himself with all his ardour to poetry and the arts, in which he also excelled. He appears to have been a sincere friend and much beloved by his students; his loss, after a brief attack of pneumonia, is deplored by all classes in his own country. Our hearty sympathy unites with theirs in revering his memory.

The first Fellow who was called away from among us during the present year was one well known and appreciated by your seniors, but who, owing to advancing age and physical incapacity, has not been seen among us for fifteen years. *Alexander Shaw, F.R.C.S.*, died in his eighty-sixth year on January 18th. Eminent as a surgeon and as a contributor to medical literature, his special claim to be remembered with gratitude by his professional brethren lies in the fact that his elder brother John and he[1] assisted Sir Charles Bell, subsequently their brother-in-law, in carrying out the experiments which constituted Bell the founder of modern neurology. Having been educated in his profession at the Great Windmill Street School and at Middlesex Hospital, Shaw's first work, entitled A Narration of the Discoveries of Sir Charles Bell in the Nervous System, appeared in 1828, eleven years after he had become a Member of the Royal College of Surgeons, and gave a full account of Sir Charles Bell's experiments and conclusions. Although already Galen had asserted that there were distinct nerves for the functions of motion and sensation, the actual demonstration of the existence of these two classes of nerves was given, to the satisfaction of the medical profession, for the first time by Sir Charles; and Mr. Shaw disposes, I think conclusively, of any claims that were raised in behalf of Majendie and others to the priority of discovery.

[1] Mr. John Shaw had been for fourteen years Sir Charles's pupil and assistant when he wrote his paper On Partial Paralysis. I find no evidence of Mr. Alexander's having occupied quite as important a position. Sir C. Bell's essay On the Anatomy of the Brain, in which he first announces his discovery of the nerves of motion and sensation, was published in 1811. See John Shaw's paper in Med.-Chir. Transactions, 1822.)

Mr. Alexander Shaw became a Fellow of this Society in 1836, and had therefore been connected with it for nearly fifty-three years when he died. He contributed four papers to the Transactions,[1] and successively held the offices of Councillor, Secretary, Vice-President, and Treasurer, besides serving as a member of the Library Committee, and repeatedly during his long career as Referee. One who is well able to judge states that Mr. Alexander Shaw's contributions to our knowledge of rickets form an indispensable part of the classics of that subject, which were afterwards embodied in a valuable article in Holmes' System of Surgery. Mr. Shaw, though an able surgeon and an eminently studious man, was not a voluminous writer. His taste was fastidious in the extreme, and his self-criticism severe, so that his corrections were often as voluminous as the original manuscript; but the value of his work was proportioned to the care with which it was produced. He long formed one of the ornaments of the school of Middlesex Hospital, where he had the reputation of having even higher qualities than his public career indicated.

His accomplished brother John died early, but a sister survives at the age of ninety-one, and two of his brothers died very recently at an advanced age. His declining years were soothed by the affection of his wife, who survives him, but his only child died young.

Those who were honoured by Mr. Shaw's friendship were always welcome to him, and had an opportunity of seeing how happy a good man may be in his decline, and how the memory of a well-spent life can light up the dreary hours of old age and infirmity.[2]

[1] The following are Alexander Shaw's contributions to the Transactions:—On a Peculiarity in the Conformation of the Skeleton in Rickets, vol. xvii, 434; On the Effect of Rickets upon the Growth of the Skull, vol. xxvi, 336; Description of a Specimen of Dislocation of the Atlas upon the Vertebra Dentata, attended with Contraction and Distortion of the Vertebral Canal, vol. xxxi, 289; Case of Popliteal Aneurism successfully treated by Continued Flexion of the Knee-joint, vol. xlii, 209.

[2] Mr. Alexander Shaw came of a long-lived race well known in Ayrshire;

The estimation in which *Sir William Gull, Bart.*, who is the next Fellow who has recently ended his earthly career, was held by the general as well as the professional public, has been more emphatically shown by the tributes paid to him in the press than I remember to have seen under similar circumstances. There was much, both in the man and in the course he ran, to fascinate and to command homage. Great natural endowments, combined with energy and perseverance in all he undertook, raised Gull to the high position he for many years occupied in the medical profession.

He was born at St. Leonard's, Colchester, on December 21st, 1816, and died, after an illness of above two years' duration, on the 29th January, 1890. Owing to the limited means of a widowed mother—the father having died when the subject of this brief memoir was ten years old—his school education was of a scanty kind, and, like many other men who have risen to eminence, he attributed much of his after success to the training he received from his mother, who is stated to have been endowed with great intelligence. The flow of the tide which carried him eventually to the pinnacle he attained, commenced when the then all-powerful Treasurer of Guy's Hospital, Mr. Benjamin Harrison, paid a visit to some hospital property in Essex, where he made the acquaintance of young Gull, and was struck by his activity and innate politeness. Finding that he was usher in a village school, and that he bore a high character in the locality, he induced him to come to Guy's Hospital, where in the first instance he assisted the apothecary, Mr. James Stocker, at a salary

his grandfather, David, was for sixty years minister of Coylton, in that county, and his great-grandfather for fifty-two years minister of Edenkillie, in Morayshire. Mr. Alexander Shaw's father, Charles, was for many years clerk to the Justices of the Peace for Ayr, an office which has just passed through his son and his grandson to another David. Many of our deceased Fellow's near relatives distinguished themselves in various walks of life. He himself, after studying at Glasgow, proceeded to Downing College, Cambridge, but left in 1827 before attaining his degree, in order to take the place of his deceased brother John as assistant to Sir C. Bell.

of £1 a week, in making up the medicines. Living accommodation was found for him in a couple of rooms, occupied now by bathmen, where he had a daily chop prepared for him by the midwife, then in partial charge of the maternity department.

It is unnecessary for me to follow further the gradual development of Gull's career at Guy's Hospital Whatever he did was to his credit, and aided in his gradual but sure advancement. In 1841 he graduated as M.B. at the University of London, and in 1846 the same University, conferred upon him the full degree of M.D., with a gold medal for a commentary on a case in medicine.

As a teacher at Guy's of physiology and clinical medicine he is said to have been earnest to enthusiasm; and my informant, for many years connected with the hospital, states that the students, to a man, adored him. One point in connection with Guy's that has not been mentioned in the many biographies that have been devoted to Gull is, that he is the only physician to the Hospital who has ever been appointed one of the Governors, an honour conferred upon him in 1887.

Long previously successful in drawing patients to his consulting-room, he was summoned in 1871 to attend H.R.H. the Prince of Wales in a severe attack of typhoid; and Sir William Jenner and Gull were successful in carrying the royal patient through all its phases to complete recovery; in reward for this the latter was created a baronet;[1] until his paralytic seizure in 1887 he was probably as much, if not more sought after as a physician than any other Fellow of the Royal College of Physicians has been.

Sir William was elected a Fellow of this Society in 1849, he was a member of Council in 1864, Vice-President in 1874, Referee from 1855 to 1863, and he contributed four papers to the Transactions.[2] An important paper of

[1] Sir William Jenner on the same occasion received the dignity of K.C.B.

[2] The following are the titles of Gull's contributions to the Transactions:—Cases of Phlebitis with Pneumonia and Pleurisy from Chronic Disease of

his on acquired cretinism entitled, On a Cretinoid State supervening in Adult Life in Women, is to be found in the Transactions of the Clinical Society.[1] The Fellowship of the Royal College of Physicians was conferred upon Gull in 1848; he filled the offices of junior and senior Censor, and was several times a member of the Council. In 1849 he delivered the Goulstonian, and in 1870 the Harveian Lectures. In 1854, in conjunction with the late Dr. Baly, he published, under the direction of the Royal College of Physicians, a voluminous and comprehensive report on Cholera.

Time would not allow me to enter more fully into the details of Sir William Gull's remarkable career, and to estimate the influence he has exercised on the profession. We have all known him, and the data to enable you to form your judgment have in one form or another been placed before you. Sir William Gull leaves a widow, the daughter of Colonel Lacy, to whom he was married in 1848, one son, and a daughter to mourn his loss.

In the necrology of last year four names escaped my notice, to which I must ask your permission to revert: they are those of *Dr. Robert M'Donnell, F.R.S.*, Surgeon to Steevens and Jervis Street Hospitals, Dublin, who became a Fellow in 1862, and contributed two papers to our Transactions ;[2] the date of his decease was May 6th, 1889, when he was sixty-one years old; *Dr. John Crockett Fish*, whose Fellowship dates from 1866, and who died on June

the Ear, vol. xxxviii, p. 157; Cases of Paraplegia associated with Gonorrhœa and Stricture of the Urethra, vol. xxxix, p. 195; Remarks on the Natural History of Rheumatic Fever, by W. W. Gull, M.D., and H. G. Sutton, M.B., vol. lii, p. 43; On the Pathology of the Morbid State commonly called Chronic Bright's Disease with Contracted Kidney, 'Arterio-capillary Fibrosis,' by Sir W. Gull, M.D., and H. G. Sutton, M.B., vol. lv, p. 273.

[1] Transactions of the Clinical Society, vol. vii, p. 180.

[2] The titles of Dr. M'Donnell's papers in the Transactions are—Observations on S. Gordon's Case in which Trephining of the Spine was performed, vol. xlix, p. 21; On a Case of Double Facial Palsy, with Observations on the Physiology of the Nerves supplying the Forepart of the Tongue, vol. lviii, p. 369.

29th last, at the age of fifty-four; *Dr. John Edmund Currey*, who died, aged seventy, on July 15th last, and whose Fellowship dated from 1847; and *Dr. Thomas Alexander Wise*, whose Fellowship dates from the Lincoln's Inn phase of our Society, he having been elected to the Fellowship in 1825; he quitted this life at the mature age of eighty-eight, on the 23rd of July, 1889.

Dr. M'Donnell graduated at Dublin, and served as a civil surgeon in the Crimean war, receiving the thanks of his superior for his devotion to duty. On his return he was appointed teacher of anatomy and physiology at the Richmond School of Medicine, and in 1866 was elected Surgeon to Steevens Hospital. He was considered a most remarkable man, and enjoyed the approbation of his *confrères*. He was twice married, first to Miss Molloy, and secondly to Miss M'Causland, by whom he had one son, who survives.

Dr. Wise graduated in Edinburgh in 1824, and entered the Bengal Medical Service in 1827; after a long period of service at Dacca as civil surgeon, where he showed much ability and was greatly respected, he left India in 1851, and spent many years in retirement at Norwood. He wrote a very learned Commentary on the Hindu System of Medicine, which was published in Calcutta in 1845; and he occupied the evening of his life in antiquarian researches, which would possess special attractions for those speculative historians who deal with the mythical ages.[1]

The ordinary work of our Society has been carried on during the past year, in spite of numerous difficulties, with the same zeal as ever, and our numbers show the increasing appreciation that prevails in the profession of the many advantages offered by the Royal Medical and Chirurgical

[1] The following is the title of Dr. Wise's last work:—History of Paganism in Caledonia, with an Examination into the Influence of Asiatic Philosophy and the Gradual Development of Christianity in Pictavia. By Thos. A. Wise, M.D., F.R.S.E., F.R.A.S., F.S.A.Scot., &c. London, 1885. Pp. 259. Largely illustrated. It should be studied in connection with Du Chaillu's recent work on the Viking Age.

Society. It is no small credit to our secretaries and our resident librarian that, although the resolution of last March the 4th necessitated for many months the subversion of all our library and other arrangements, they have succeeded in bringing out the eighty-second volume of the Transactions in as satisfactory a condition as any of its predecessors. Our thanks are specially due to them for this and much else that they have done in the service of the Society; for if they had followed the precedent of 1834, when the Society moved from Lincoln's Inn to Berners Street, no Transactions would have been issued. I have on a former occasion, when we first met in this house, dwelt on the labours of the secretaries and of the Building Committee; and you, though you can scarcely know all the difficulties and disappointments they have experienced, see before you the magnificent result of the decision at which you arrived shortly after the last Annual General Meeting.

Among the various acts which our new birth had given rise to is one that specially deserves to be signalised on this occasion, the more so as it may serve as an example to be followed; it is the establishment of an Endowment Fund, which was considered by the Council as the best use to which £50 presented to the Society by Dr. Quain could be put. This has already been increased by further donations, and constitutes a nucleus which will, I trust, before the jubilee of our last migration is celebrated, be augmented by many thousands. For it is not to be supposed that we are rich, or that good work can be done without cost. We are already taxed individually to an enormous extent, in the shape of gratuitous work performed for the community. It is not just that we as a Society, should ask our Fellows to devote their scant leisure to the advancement of science at a pecuniary cost to themselves. And if our scientific committees are to carry out their investigations to a satisfactory conclusion, the least we can do is to facilitate by all legitimate means, the researches that involve not only the exercise of much brain-power, but a considerable pecuniary outlay. This

is one impediment to the appointment of Scientific Committees—of which one, that on British Climatology and Balneology, has been most zealously labouring since it was appointed, and some of the results of their inquiries and observations will doubtless soon be brought before you. This committee involves not only a large correspondence, but an outlay for travelling and for scientific instruments which our present cramped means have some difficulty in meeting. Another scientific committee has been much talked of, and is required to supplement and correct the report of the former Committee on Suspended Animation, but the *res angusta domi* has not as yet allowed it to come into operation. May the new Council see its way to complete the inquiry, which we are almost pledged to carry out, and which the lapse of twenty-eight years[1] renders a great desideratum on account of the practical issues involved.

Our credit as a scientific society depends on the work we continue to perform; and with the increase of knowledge an increasing demand will be made upon us and our successors, which dare not be ignored except at a sacrifice of the high position established by our predecessors. "Science," to use Professor Bunge's words, "will continue to ask and to answer ever bolder questions. Nothing can stop its victorious career, not even the limitation of our intellect. This, too, is capable of being made more perfect. There is no rational ground for thinking that the continuous progression, development, and ennoblement of type which has been going on for centuries on this planet should come to an end with us. There was a time when the only living creatures were the infusoria floating in the primeval sea; and the time may come when a race will dominate the globe as superior to ourselves in intellectual faculties as we are to the infusoria."

Fellows of the Royal Medical and Chirurgical Society,

[1] See Report of the Committee appointed to investigate the Subject of Suspended Animation, Transactions, vol. xlv, 1862, p. 249.

my faith in our future is firm. We individuals pass away; but though

"The old order changeth, yielding place to new,"

we may be assured that there will be a certain harvest where a good seed has been sown; it is our duty to see that the grand legacy we have received from our predecessors is handed down to our descendants not only undiminished, but strengthened, increased, and beautified.

With what words can I better close this address than with those of thanks to you who have placed me in this chair, and have for two years leniently and kindly borne with my shortcomings? How can I adequately express, on my retirement from office, my gratitude to all the officials with whom I have been brought into contact? I will not detain you with any emotional remarks, but you will, I am sure, pardon me if I congratulate you on your choice of my successor, eminent alike as a writer and as a surgeon, and especially qualified by his long services to this Society, and his intimate knowledge of all the processes connected with its new birth, to direct its further growth and development. I particularly congratulate you upon the retention of your excellent treasurers, Dr. Hare and Mr. Bostock, of your indefatigable secretaries, Dr. Taylor and Mr. Warrington Haward, and of your learned librarians, Dr. Gee and Mr. Hulke; by their aid, and that of the distinguished members of the new Council, under the guidance of Mr. Timothy Holmes, I can have no hesitation in prognosticating the continued progress of the Royal Medical and Chirurgical Society. I cannot add anything more forcible in praise of the resident librarian, Mr. Mac Alister, to what has been said in the report of the Council; but I am confident that I am only echoing your sentiments if I express a hope that in his new surroundings he may find the reward for the great anxiety and the labours that our migration has entailed upon him.

Fellows of the Royal Medical and Chirurgical Society, I bid you farewell.

AN ANALYTICAL AND CLINICAL EXAMINATION

OF

LEAD-POISONING IN ITS ACUTE MANIFESTATIONS.

BY

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Newcastle-upon-Tyne and the surrounding district have been for long the home of the lead trade. At the present time the amount of lead and silver ore raised in the counties of Northumberland, Durham, and Cumberland is considerably less than formerly, but this is counterbalanced by the very large imports into the Tyne of a richer lead ore from Spain, and thus it is that there is greater activity in the lead industries to-day than there ever has been. As an illustration, in the year 1862 the amount of white-lead alone manufactured was 7500 tons, and in 1887 it was 14,000 tons.

Of all the industries on Tyneside, lead-making is the one which has unfortunately gained for itself a bad name. Lead workers are not as a rule a healthy class of people. Too soon, in spite of precautions which, it must

be admitted, are not always attended to by the workpeople, many of those who are engaged in the process become indisposed. It is the workers amongst *white*-lead rather than *red*-lead that suffer the more frequently and severely, although the lead-smelter and separator of the ore may suffer. No local industry sends to the Newcastle Infirmary such human wrecks as lead-works do. On looking over our Infirmary Registers for the last five years I find that 135 cases of lead-poisoning were admitted as in-patients. Of these, ninety-one were women, and forty-four were men; eight died, three men and five women. Most of the women were young, and died soon after being exposed to the influence of lead. The eight deaths reported do not represent the total number of deaths from the effects of lead-poisoning, but only those who died from the immediate or primary effects of lead.

The danger to the individual from the inhalation of lead begins with the process of smelting. The lead miner never suffers. Animals that graze in the neighbourhood where lead-smelting is carried on suffer from colic and other symptoms of lead-poisoning. In Weardale this fact has long been known to the farmers who from time to time have received compensation for the injury thus inflicted upon their cattle. Within the last few years a hood has been placed in front of the furnace in the lead-mills, and since then smelters have not suffered. I have known smelters suffer most severely from the effects of lead-poisoning. In one case, to my knowledge, four sons in one family, all stalwart men, died from the effects of lead-poisoning through the development of kidney disease—a circumstance which, supported by other experience, makes me believe in the existence of not only an individual, but a family predisposition to plumbism.

The worst effects of lead are met with amongst the *white*-lead workers. Women and girls suffer not only much more severely, but much earlier from the effects of lead than men. In a few days in some instances, or at the most after a few weeks' exposure to lead, either in

what is known as the "white beds" or the stoves, there is produced an anæmia which goes on rapidly increasing. Colic and headache are complained of, and occasionally vomiting and disturbances of sight, amongst which I would mention double vision and amaurosis.

The excellent system which prevails at our lead-works of a weekly inspection of the workers by medical men is one of the best preventives of lead-impregnation that I know of. The slightest indication of lead-contamination noticed by the medical examiner is made the occasion of a recommendation to the employers for a three months' suspension from labour on the part of the lead-workers. Knowledge of this fact, however, not only causes the work-people not to complain when inspected by the doctor, but to insist upon feeling quite well, although an hour or two after the inspection they may be found suffering from colic and unable to follow their occupation. The high wages tempt them to deceive not only the doctor, but also the employer. Lists of the workpeople who are suspended on account of illness are circulated amongst the lead manufacturers of the district. A woman suspended at one place, having as she thinks recovered her health and unwilling to be idle for three months, applies at another factory in the district for employment, using a false name. It is only by the most careful discrimination on the part of the manager and examination on the part of the doctor that this rule is not more widely broken. It was on looking over the lists in the factories of workpeople who have been suspended, when I have so very frequently seen the names of girls who were sisters or cousins to each other, all of whom had suffered from lead-poisoning, that I have been led to believe in the existence of a family predisposition to plumbism.

In spite of all the precautions possible that are taken by the employers to prevent contamination of the work-people, the fact remains that every now and then a girl of from eighteen to twenty-three years of age works only a few weeks or months in a lead factory when symptoms of

acute lead-poisoning are noticed, namely, colic, constipation, vomiting, headache, pains in the limbs, and incomplete blindness. In a few days, with or without treatment, she becomes convulsed, and dies in a state of coma, the death being so sudden that we cannot but regard it as due to an acute toxæmia, and in some way or other dependent upon the influence of lead. In most of these cases albuminuria is absent, and at the post-mortem no organic change is found save a hydræmic and anæmic condition of the brain; and on chemical analysis, as shown in the charts, lead is found in the various organs, *e. g.* the brain, liver, and kidneys.

It is not so much my wish to give in detail the physical signs and symptoms of lead-poisoning as to draw attention to some peculiarities connected with them, and above all to the pathology of the acute cases.

The presence of a blue line on the gums is a physical sign of very great importance when present. In some of my cases, however, it has been absent, and yet the patients have suffered not only from colic, but from symptoms of lead encephalopathy. In a paper published in the 'Brit. Med. Journal' for October, 1885, I stated that I had found a blue line present in 13 out of 18 cases—or in 72 per cent. A few weeks ago I visited one of the lead factories in my neighbourhood, and examined 38 women of all ages from eighteen to seventy-two, taken at random and engaged in various departments, and I found a blue line present in 28 out of these 38, or in other words 73 per cent. As these two numbers almost tally, I take it that this is about the usual percentage. It is a sign not always to be relied upon; as we have seen, it may be absent, when other symptoms of poisoning are present. I have seen the blue line well marked in girls who have worked only one week in the factory. Here, however, we must be careful to distinguish between a blue line due to the deposition of sulphide of lead in the gum and the discoloration which occurs by the simple deposit of lead dust on the surface of the gum seen in girls who have been only a day or two in

the factory. The latter easily disappears after washing and cleansing the mouth. That the blue line is due to the action of sulphuretted hydrogen upon lead circulating in the blood is confirmed by a circumstance such as this, that in one of my patients—not a lead-worker—two drachms of acetate of lead were taken with suicidal intent, and on the following day a distinct blue line had developed in the gum, which persisted for several days. Dr. Inglis, of Jarrow, has found in many old lead-workers, in addition to the blue line, dark discoloured patches inside the lip opposite ragged canine teeth; these patches are as a rule irregular in shape, and seem to depend upon blocking of the follicular glands by particles of lead. In one of his cases presenting this sign the woman has worked almost continuously for seven years without suffering. The same observer also tells me that he has frequently met with dark blue lines and stains in the middle portion of the small intestine, and with large patches of staining in the large intestine. The presence of these patches is with difficulty explained, unless it be that they are dependent upon hardened pieces of fæcal matter strongly impregnated with lead having lain for a considerable time there, so as to allow of absorption taking place. The stain, it is to be remembered, is beneath the mucous membrane.

Of such signs as wrist-drop and paralysis I shall say nothing, save that the paralysis occasionally extends to muscles of the arm other than those supplied by the musculo-spiral nerve: these all undergo atrophy. I have seen the peroneal muscles affected.

Nor of colic shall I say anything except that it is a most common symptom, frequently obliging the lead-worker to desist from his or her occupation whilst no other indication of poisoning is present. It is difficult to explain this colic. The metal is undoubtedly present in the tissues, but, as will be seen on referring to the table which deals with the amounts of lead found in the various tissues after death, the quantity found in the intestine is small. I have never found the wall of the intestine thickened, as

some writers maintain; nor will a general ischæmia explain the colic, though a partial ischæmia may. The pain is dependent upon muscular spasm of the intestinal wall.[1] Knowing the tendency of the nervous system to become rapidly affected by lead, and some parts more quickly and profoundly than others, it is just possible that the abdominal sympathetic ganglia are variably affected; some more than others, and some, perhaps, not at all. The result, then, would be incomplete paralysis of some segments of the intestine and over-action of others.

People who have been long exposed to the influence of lead suffer as time goes on from renal disease—followed, it may be, by disease of the heart; and death comes either from albuminuria being followed by exhaustion and allowing of the development of some intercurrent affection, or from uræmia or cerebral hæmorrhage.

But I would call attention especially to certain cases of acute lead-poisoning. For example, a girl works, it may be, only a few weeks or months in a lead factory, when, after having been noticed by her friends to have been rapidly becoming anæmic, she complains of colic, constipation, headache, dimness of vision, and in a few days afterwards develops convulsions, or becomes delirious and dies comatose. As the symptoms are so rapidly developed, and as no organic change is found post mortem, the death can only be attributed to toxæmia. Death in these cases is analogous to death from strychnine-poisoning. From the hydræmic condition of the brain found after death, the inference is that there has been irritation of the vaso-motor centre, and spasm of the cerebral arteries; and that these conditions are caused either by the lead itself acting as a poison to the nervous system,

[1] To this extent I follow Harnack, who also states that the colic is due to irritation of the intestinal ganglia. In man, he says, the result of this irritation is a spasmodic contraction of the intestine producing constipation; whilst in animals, we are told, the same cause produces increased peristalsis and diarrhœa. I find in this an explanation of the increased general arterial tension which may be present, but not of the colic which is always more or less localised.

or by the poisoning of the blood from retention and circulation of effete material due to lead interfering with the function of the emunctories.

When we come to analyse the symptoms in these cases, what we find is that in them as in the less acute cases, and my remarks now will apply to both, there have been colic, vomiting, headache, and constipation, and that for some time past the patient has been very anæmic. There is no doubt about the anæmia, or saturnine cachexia, as it is called: hæmocytometric observations show a very marked disappearance of red, and a slight increase of white, blood-corpuscles. What the anæmia is due to is another thing,—one, too, very difficult to explain. All the women engaged in this industry suffer sooner or later from disordered menstruation: young girls at first from excessive menstruation, and married women also from menorrhagia; these exhibit a marked tendency to abort if pregnant; others suffer from amenorrhœa. In exactly one half of the women questioned at the factory, between the ages of eighteen and forty-five, the menses were excessive; in one menstruation had been for years suppressed; in the rest it was regular. We may therefore find a partial explanation of the anæmia in this excessive menstruation, which I certainly regard as one of the peculiar and pernicious influences of lead upon women; and it is just possible that in this disordered and excessive menstruation lies the secret of women suffering more than men from lead-poisoning. But one half of the women have regular menstruation, and are yet anæmic: men, too, suffer from this cachexia. We are therefore obliged to admit that lead exercises a very prejudicial effect upon the blood itself or upon the blood-making organs.

Accompanying the headache is dimness of vision. Two or three of my patients had diplopia. In these cases where vision is obscured the most marked optic neuritis is found, and this at a time when, as no albumen is present in the urine, the change in structure must be due to some peculiar influence of lead upon nerve. The signs are those

of ordinary optic neuritis. This is very quickly followed by atrophy in some instances, and permanent blindness may be left: in many, on the other hand, the optic neuritis quite clears away. In all, however, it is an indication of the severity of the lesion and a measure of the danger. Later on, when albuminuria has been developed, there have been superadded to the physical signs of lead neuritis those frequently noticed in kidney disease.

The brain symptoms are such as we might expect in toxæmia, viz. headache, vomiting, delirium, convulsions, and coma. In one of my cases there was right hemiplegia with aphasia; in another the most marked tremor of arm and leg. As a symptom aphasia has not been much noticed. Dr. Inglis, whose experience amongst lead-workers is great, met with one case of aphasia which was followed by eclampsia. The aphasia lasted for nine months, and was accompanied by agraphia. Speech returned, but rather imperfectly. The patient is now married, and has several healthy children. In this case eclampsia and aphasia occurred without there being albumen in the urine. The tendency is, however, for the kidneys to become affected as time goes on. The organs are small as a rule, and microscopical examination shows a marked increase of the interstitial tissue.

Although in nearly all our cases of lead-poisoning in Newcastle the kidneys are found to be contracted, and resembling the gouty kidney, though not so red, gout is practically unknown amongst our lead-workers. In only a very few instances have I met with rheumatic arthritis, and in only two have I met with gout, and this was in the case of a young girl who was a lead-worker, and whose father and mother had also both worked in the lead factory and had suffered. The absence of gout in our neighbourhood amongst lead-workers is a subject of more than passing interest, it is one of great physiological importance. I have discussed it with many of the London physicians who have come to Newcastle as Examiners in Medicine for the University of Durham, and with no satis-

factory explanation. They see the association of gout and lead so frequently in London that they are forced to admit the relationship. In the treatise on gout recently published by Sir Dyce Duckworth the subject is discussed at considerable length. His own experience, as well as that of others, is given; amongst which is the interesting case of Dr. Lauder Brunton, where a few grains of lead and opium pill given for diarrhœa to a painter previously healthy were followed in a few days afterwards by a distinct development of gout. Opposed to this relationship of gout and lead is the testimony given by many physicians of provincial and Scottish hospitals. Amongst the former is the opinion of my colleague Dr. Drummond, who also states that in the north this relationship is never noticed. Our opinion is that in the north of England gout is practically unknown as a symptom of lead-poisoning; it is the last symptom I should either look for or expect to find. It is to be remembered, however, that gout, generally speaking, is not a common disease with us. I have tried to find an explanation of the absence of gout amongst our lead-workers, but have hitherto failed. I do not think it is altogether a question of malt liquors being drunk by the London workmen, and of whisky by those in the north. What it is I do not at present know. Climatic conditions may have an influence. What we believe is that lead in some way or other so influences the metabolism of the tissues that the ordinary nitrogenous waste is either improperly formed or imperfectly eliminated. In most of my cases there has been a marked diminution in the daily discharge of urea, 200 to 250 grains being the average: in some of the cases under treatment the amount rose to near the normal, whilst in others it diminished. It is to the amounts of uric acid eliminated daily that I would invite attention. Here I admit we are dealing with a difficult subject, since we do not know definitely what diurnal variations of uric acid elimination are consistent with health, but it is upon this point that almost everything centres so far as the development of gout is concerned. Physiologists give

varying amounts for the daily discharge of uric acid; Flint says 6—9 grains, McKendrick 13 grains, Brubaker 8 grains, Kirkes 8·5 grains, Landois 7—10 grains, Ralfe 7 grains, and Foster 7—8 grains.

Now of four cases the details of which were worked out for me by Dr. Bedson, Professor of Chemistry in the Durham College of Science, the following are, roughly speaking, some of the average eliminations.

McNay—for some days before treatment the average quantity was 5·9 grains, and after treatment the average of several days was 7·7 grains.

Miller—before treatment 16·7 grains, after treatment 8·4 grains.

Buglas—before treatment 12·5 grains, after treatment 14·57 grains.

Ruddy—before treatment 7·7 grains, after treatment 7·1 grains.

The methods used by Professor Bedson were for urea the hypobromite of soda; for uric acid Haycraft's method; and for estimating the amount of lead in the urine and tissues the colorimetric test.

In only one of these cases, therefore, was the daily discharge of uric acid below the amount stated by physiologists as the normal. Admitting the correctness of the uric acid theory of gout, there were not in existence in these cases the conditions that lead up to the development of gout, unless circumstances arose to check the elimination of uric acid. On looking again at the charts it will be seen that the daily discharge of uric acid was occasionally twice, sometimes thrice, what it ought to have been. Lead has therefore some peculiar influence upon the formation of uric acid in the system.

Another interesting point is that lead was found daily in the urine of patients under observation; and that under treatment by potassium iodide the amount of lead thrown out daily by this channel increased in quantity. In nearly every case the amount of lead discharged was doubled or trebled.

In Buglas's case the amount of lead for several days at first was ·0126 grain; it rose to ·334 grain.

Ruddy, from ·0208 to ·0297 parts of a grain.

Miller, from ·042 to ·073 parts of a grain.

McNay, from ·0035 to ·0301.

The charts also show another interesting point as regards these eliminations: every now and then there seems to have been a kind of explosive elimination of lead and uric acid, and it would appear as if they stood in an inverted relationship to each other. Nothing occurred to predict these sudden rises and falls, nor was anything noticed to follow them.

The other tables show that a very small amount of lead is met with in the tissues after death; that a small amount after all has been absorbed, and yet has been capable of causing death. Less than one grain of lead found in the brain after death! this seems a small amount to have caused such terrible suffering and an early death! True, it represents metallic lead, and we know nothing of how it is combined with the tissues, or even in what chemical form it exists, although there is much to lead us to infer that it exists in some peculiarly complex molecular form. Still I cannot but think that the death is from acute toxæmia, analogous to but not identical with uræmia; the individual is poisoned by the products of her own metabolism; her cachexia points to a rapid disintegration of blood, and the presence of lead in the bones and spleen after death lends weight to the opinion that a deep wound has been inflicted upon the blood and blood-making organs.

Can nothing be done to diminish this tendency to lead-poisoning and rapid death? The blame, I admit, is not altogether due to lead-making. There is an individual predisposition to plumbism. There is a class of women too easily affected by lead, but what that type is it is difficult to say. All I can say here is that many of them are ill-fed, badly housed, and lead a questionable life, and thus, owing to starvation and exposure, may be regarded

as subjects likely to break down quickly under the influence of lead. But to many these remarks will not apply, and consequently these conditions, whilst predisposing or tending to aggravate symptoms when present, cannot be the cause of them.

I would summarize my opinions thus:

1. That women suffer much more frequently and severely than men.

2. That women suffer at an earlier age than men: that, for example, of the 135 patients admitted into the Newcastle Infirmary, whilst up to the age of twenty-three no men were affected, forty-nine women had already suffered; that after the middle term of life men suffer more than women.

3. That acute lead-poisoning attended by cerebral symptoms is much more fatal amongst women than men.

4. That the most fatal period of lead-poisoning is that time in a woman's life when the menstrual function is extremely active, that this is one of the functions of the body most apt to be quickly disturbed, and that in this way an explanation may be found of the greater prevalence of lead-poisoning amongst women.

5. That death in the acute stage is due to toxæmia, and in chronic plumbism is due to organic changes in the kidneys and nervous system.

6. That gout in the north of England is a very infrequent accompaniment of lead-poisoning.

7. That cardio-renal changes are the most frequent consequences of slowly developed lead-poisoning. That whilst the paralysis known as "wrist-drop" is more frequently met with amongst men than women, women suffer much more frequently from the acute cerebral symptoms.

Cases.

Case 1. *Sudden death in lead-poisoning.*—Elizabeth Ann T—, æt. 22, single, admitted into the Newcastle Infirmary July 18th, 1889; worked two and a half years at the "white-lead." After the first three months she was obliged to leave off work for three weeks, owing to colic. She returned and worked for seven weeks, when she was again obliged to leave on account of colic. In August, 1888, she had severe pain in the head, which was followed by partial blindness. She did not return to the factory for two months. Gradually she regained her eyesight, and has since then worked off and on at the lead-works. She began to menstruate at the age of fifteen; her menses, which have been regular, have been scanty since she went to the lead factory. At present the patient is menstruating. For the last few days she has complained of pain in her joints and loss of eyesight. Urine normal, free from albumen. The patient died in a convulsion early on the morning following the day on which she was admitted. The history of the case was obtained from the mother after the death of the patient.

Post-mortem.—Body that of a well-developed female. Blue line on gums. No œdema. Pupils half dilated. Lungs healthy. Pericardium healthy, contains about two drachms of serum. Heart healthy, weighs 10½ oz. Right ventricle—walls flaccid, cavity empty. Left ventricle—wall fairly thick, chamber empty, aortic valve competent. Endocardium healthy, valves all healthy. Liver smooth, healthy, weighs 60½ oz. Gall-bladder contains fluid bile in small quantity. Liver-tissue on section is seen to be pale. Spleen tears easily, is soft and pulpy, weighs 6½ oz. Left kidney 5¼ oz. Capsule is removed with ease. On section the veins in cortex and medulla are seen to be injected; otherwise nothing abnormal is detected. Right kidney 5¼ oz. Capsule removed with ease. A small quantity of pus is seen exuding from pelvis of kidney, but the lining mem-

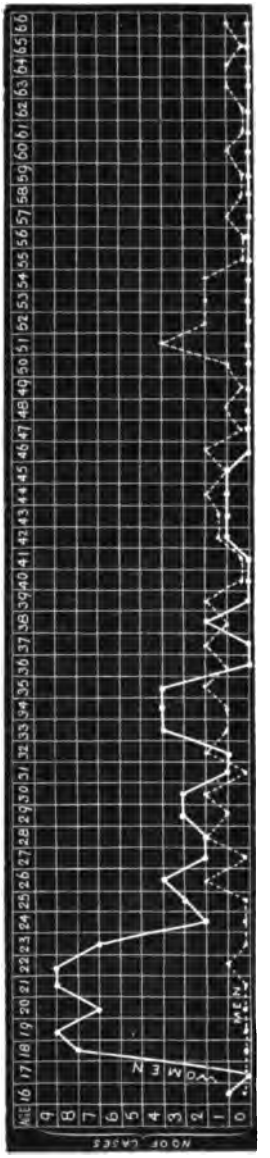
brane is not noticed to be injected. Kidney substance rather injected, but healthy. Vagina—hymen absent. Uterus—cervix eroded and granular. Interior of uterus covered with a red slimy material, which may be menstrual. Ovaries—right ovary enlarged, contains two or three corpora lutea, one yellowish, the others rather red, but evi-

Name of organ.	Total lead in parts per million.	Weight of organ.	Grains of lead per weight of organ.
Lung . . .	7·6	29·0 ounces	0·0964
Heart . . .	4·12	10·5 „	0·0189
Liver . . .	37·8	60·5 „	1·000
Spleen . . .	12·0	6·5 „	0·0341
Kidneys . .	10·0	5·25 „	0·0229
Cerebrum . .	9·8	51·5 „	0·779
Cerebellum . .	24·8		
Pons . . .	22·6		
Spinal cord . .	1·16		
Large intestine .	37·7		

	Alcoholic extract, lead in milligrms.	Ethereal extract, lead in milligrms.	Aqueous extract, lead in milligrms.	Ash, lead in milligrms.	Total lead in milligrms.	Lead, parts per million.
Pons . . .	0·14	0·35	0·0	6·1	0·59	22·6
Cerebellum . .	0·25	0·4	0·0	1·15	1·80	24·8
Brain . . .	0·3	0·0	0·0	1·35	1·65	9·8

dently not recent; left ovary smaller, and somewhat cystic. Stomach healthy, small ecchymoses near pylorus in upper wall. Large intestine—longitudinal and circular muscular fibres well developed, mucous membrane distinctly injected. Brain—dura mater slightly adherent at vertex. Sub-

Lead-poisoning Cases admitted into Royal Infirmary, Newcastle-on-Tyne, during Five Years ending June, 1889.



arachnoid fluid has accumulated to excess in the interpeduncular space; pons and cerebellum extremely pale compared with rest of brain, the pallor being particularly noticeable in the pons. The surface of the brain is healthy; vessels not unduly injected. Corpus callosum very pale, as also brain-tissue generally; very few puncta hæmorrhagica. Each lateral ventricle contains two or three drachms of serum. Membranes of brain other than stated above are healthy, and there is no effusion. Spinal cord feels extremely hard and is pale.

Case 2. *Rapid death in lead-poisoning.*—Catherine H—, æt. 21, single, admitted June 28th, 1889, complaining of pain in the abdomen of eight days' duration. Was always a very healthy girl. She has worked at intervals in the lead factory during the last twelve months, and has suffered thrice from colic during that period. Only three weeks ago she returned to the factory. Her present illness commenced eight days ago with pain at the vertex; her appetite became bad and her bowels constipated. On admission patient appeared to be very ill. She was extremely restless and moaned a great deal, owing to the headache and abdominal pain. She was quite conscious; was very pale. A blue line was noticed on the gums. On the same evening from 6 to 7 p.m. patient had three fits; after 7 p.m. she became quieter. It was noticed that during the fit the left arm moved most. On the following day she was comatose; the pupils were half dilated and reacted slowly to light. Knee-jerk present, slightly exaggerated on left side. Pulse 80, slow; respirations 20 per minute. Heart—first sound over mitral area prolonged, second aortic sound accentuated. She is quite insensitive to pain. Urine, removed by catheter, measured 20 oz., *not albuminous*, sp. gr. 1010. Treatment proved unavailing, the patient never regained consciousness, and she died on June 30th.

Post-mortem.—Brain weighs 48 oz.; the convolutions of both hemispheres are flattened; the veins of the membranes

are gorged with blood. On section the brain substance is seen to be pale, œdematous, and soft. There is slight excess of fluid in the lateral ventricles. Heart—cavities are empty, valves and orifices all healthy. Lungs—left, adherent at places; upper lobe œdematous, lower lobe congested: right lung, lower lobe congested. Spleen

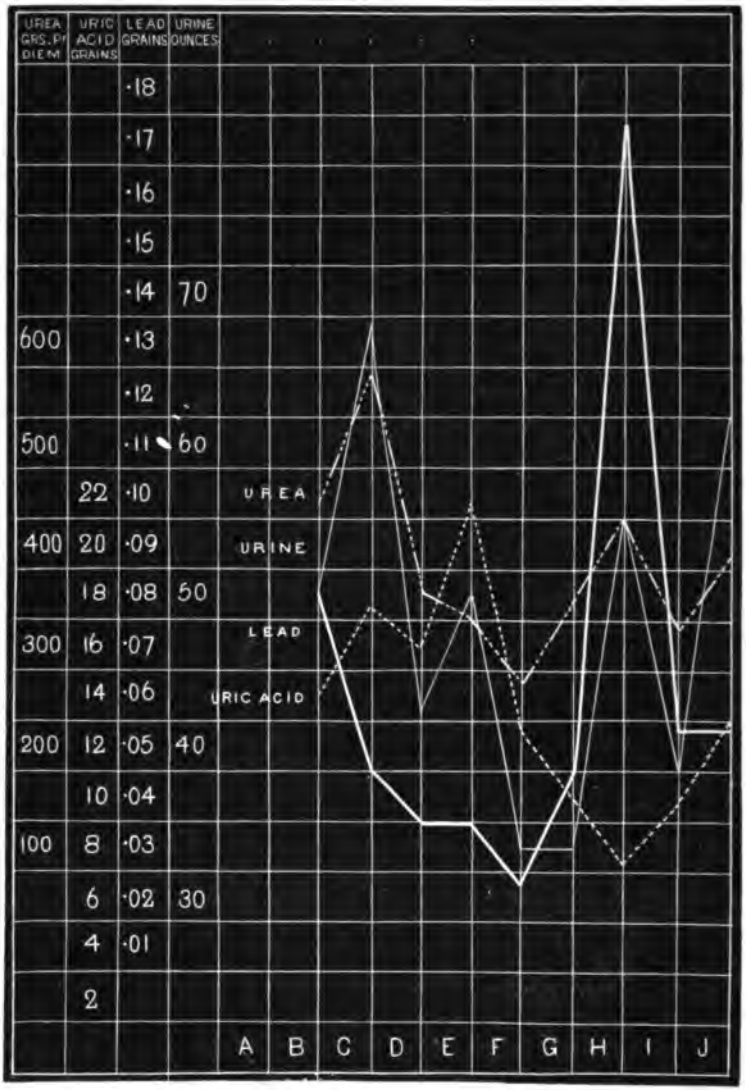
Name of organ.	Total lead in parts per million.	Weight of organ.	Grains of lead on total weight of organ.
Heart . . .	0·5	7·5 ounces	0·0016
Liver . . .	41·6	45·0 „	0·819
Kidneys . .	13·3	4·5 „	0·0261
Spleen . . .	39·0	5·0 „	0·0883
Cerebrum . .	21·6		
Cerebellum . .	8·59		
Brain with cerebellum . }	30·19	48·0 „	0·634

	Alcoholic extract, lead in milligrms.	Ethereal extract, lead in milligrms.	Aqueous extract, lead in milligrms.	Ash, lead in milligrms.	Total lead in milligrms.	Lead, parts per million.
Brain . . .	0·6	0·6	0·91	1·3	3·41	21·6

weighs 5 oz., is soft, and is studded with a large number of minute hæmorrhages. Liver weighs 40 oz.; smooth on the surface, pale on section; otherwise presents nothing abnormal. Gall-bladder contains ½ oz. of yellow bile. Kidneys—capsule is readily removed; on section they exhibit nothing abnormal. Intestine normal.

Case 3.—Mary M—, æt. 20, single, a lead-worker, admitted August 3rd, 1889, complaining of pain in the abdomen, of headache, and of vomiting. Family history good. Four years ago the patient went to the lead fac-

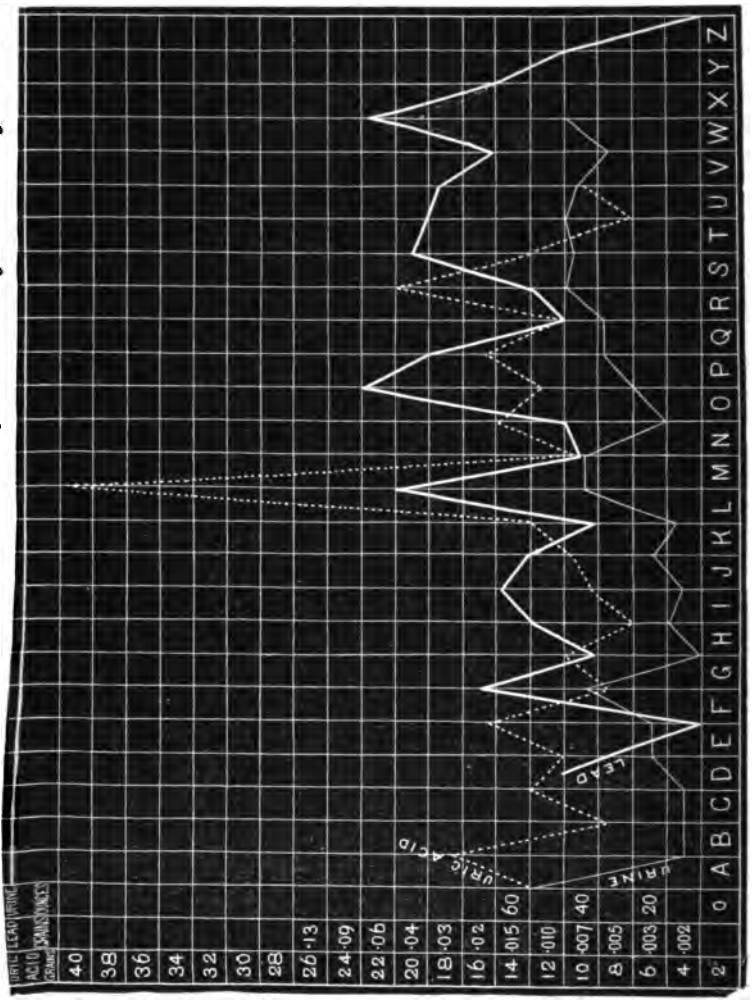
CASE 3. Mary M—.—*Daily Elimination of Urine, Urea, Uric Acid, and Lead by the Kidneys.*



tory. After working there for three months she was obliged to desist. For the next two and a half years she was employed as a hawker of fish. A year ago she returned to the lead factory, where she was employed in the "white beds and stoves." Three months afterwards she was again obliged to give up work on account of colic and headache. She was away from the factory for five months, but again returned, only, however, to suffer; for after eight weeks' employment she was again the victim of colic, headache, and vomiting. She is pale, and her features are somewhat full and rounded. Menses appeared when patient was fourteen years of age, and she continued to menstruate regularly until a year ago, when after returning to the lead-works menstruation became profuse, on two occasions the loss being so great that she could not leave the house. This was followed by amenorrhœa, which lasted three months. Since then the menses have been regular but scanty. Pulse 76, soft, compressible. Eyesight is not so good as formerly. Ophthalmoscopic examination by Mr. Williamson, August 4th: Left eye, disc woolly; large myopic crescent surrounding it; slight remnant of old choroiditis. Right eye, old choroiditis better marked here than in the other eye; disc woolly. Patient has never been diplopic, and tells us that her eyesight has never been perfect. Temperature normal. Urine 1027, no albumen, acid, 35 oz. daily average. Patient has excellent teeth, and only the faintest trace of a blue line is noticed on the gum. Tongue moist, clean. Lungs healthy. Heart, beyond slight reduplication of the first sound over the mitral area nothing abnormal is detected. There is a venous hum in the neck. In the abdomen nothing abnormal is detected. Treatment consisted principally of sulphate of magnesia with hyoscyamus and tincture of ginger, and in a fortnight all her pains had disappeared, and her eyesight had considerably improved.

Case 4.—Elizabeth B—, æt. 26, married, admitted into the Newcastle Infirmary October 30th, 1886, complaining

CASE 4.—Elizabeth B—.



Daily Elimination of Urine, Uric Acid, and Lead by the Kidneys.

of obstinate constipation, pain in the abdomen, and sickness. She is a pale anæmic woman, with rounded features; very distinct blue line on the gums; internal squint of left eye, the pupil of which is slightly more dilated at times than the right. Has been married four years; never miscarried; has never had children. Menstruation began when fourteen years of age, and has been quite regular; never had menorrhagia. Seven weeks ago she went to a lead factory, where she was engaged in the "stoves," carrying and drying white-lead. Was perfectly healthy when she went there; has since lost much of her colour. After working two weeks in the factory she had epistaxis. This continued more or less for more than half a day. Previous to this she had been losing her appetite; she was scrupulously careful in regard to washing her hands before eating, and took freely of the acid drinks provided. During the third week she began to suffer from pains in the abdomen, accompanied by distension and constipation. This was followed by vomiting every time she sat down to a meal. A disagreeable taste in her mouth too was felt, and her gums now showed the blue line. Urine alkaline, sp. gr. 1022, no albumen; contains phosphates; 60 oz. of urine passed daily on an average. Lungs healthy. Heart—first mitral sound reduplicated, otherwise the sounds are healthy. Abdomen—pain is felt over the transverse colon, relieved by pressure. Blood contains 2,620,000 corpuscles in 1 cubic mm.; there is one white corpuscle to 261 red. The patient was treated by means of bismuth, morphia, and belladonna, and made a good recovery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 11.)

A CASE OF TUBAL PREGNANCY,

WITH REMARKS ON THE CAUSE OF EARLY RUPTURE.

BY

J. BLAND SUTTON, F.R.C.S.,

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Received October 14th—Read November 12th, 1889.

It would be superfluous to occupy the time of this Society with the details of a successful operation for ruptured tubal pregnancy, were it not that the case presents some exceptional features, and enables me to offer a few remarks on some points in the pathology of the accident.

Emma B—, æt. 37, came under my care in the Middlesex Hospital, August 26th, 1889. Patient has been twice married, her matrimonial life extends over a period of seventeen years. Has never been pregnant or suffered from gonorrhœa, and has always been regular with regard to the catamenia until three months before her admission, when she missed two periods. Five weeks before admission patient was seized with sudden violent pain in the abdomen. Dr. Clegg, of Stratford, was sent for, and on

arriving found the woman collapsed. Slowly she reacted and refused to allow any vaginal examination to be made. In the course of a few days a swelling appeared on the right side of the abdomen. At the end of five weeks she was sent to me at the Middlesex Hospital.

On admission I found a swelling occupying the right iliac fossa, extending upwards to the costal arch and inwards as far as the middle line. The uterus was normal in position, and the sound entered three inches. The right side of Douglas's fossa was occupied by an ill-defined swelling, firm to the touch; a rounded moveable nodule, of the bigness of a Tangerine orange, lay behind the uterus. No breast signs or history of vomiting. There was great tenderness over the abdominal aspect of the tumour. During the next twelve days the temperature ranged from 99° in the morning to 101° in the evening.

On September 6th (twelve days after admission) I opened the abdomen, and came upon a quantity of putrid, dark-coloured blood-clot filling the pelvis and right iliac fossa, and extending upwards to the liver. This was quickly removed, as well as the rounded moveable nodule in Douglas's fossa. As soon as the clot was turned out some smart bleeding came from the right broad ligament. This was quickly stopped by transfixing the ligament with a double silk ligature close to the uterus, afterwards cutting away the débris of the tube and ovary. The cavity of the pelvis and peritoneum were washed out with eight quarts of water at 110°—115°. I then examined the left broad ligament, but the parts were so matted together that it was impossible to distinguish ovary or tube. The uterus was normal in size, shape, and position. A glass drainage-tube was inserted and retained for three days. The temperature varied from 98° to 99·4° during the six days following the operation, then rose somewhat as the track of the drainage-tube suppurated. The patient made an excellent recovery, due, I believe, to the use of the drainage-tube.

On examining the parts removed I was able to recog-

nise the remnants of an enlarged Fallopian tube, the ovary contained a corpus luteum of pregnancy. The rounded moveable mass consisted of what is known as an *apoplectic ovum*, and on washing the clot the cephalic extremity of an embryo was found, corresponding to the seventh or eighth week. An examination of the membranes is of interest, as it throws some light on the cause of these *early* ruptures in tubal pregnancies.

Obstetricians are familiar with rounded masses discharged from the uterus of pregnant women accompanied by profuse hæmorrhage. Such rounded masses are known by a variety of names—blighted ovum, carneous mole, apoplectic ovum, cystic or tubercular ovum. They are so common that every pathological museum contains many specimens. In the middle of a blighted ovum a cavity exists, usually lodging an ill-developed, misshapen embryo of about the fifth, sixth, or eighth week of pregnancy: occasionally only the stump of the cord is detected. A blighted or apoplectic ovum is an early embryo with its membranes, into which hæmorrhage has occurred. The extent of the extravasation varies; sometimes the whole of the membranes are infiltrated, and occasionally the blood invades the amniotic cavity and overwhelms the embryo.

The specimen I show to-night is an *apoplectic ovum from the Fallopian tube*, and a glance at the drawing (Fig. 1) will be sufficient to establish its identity. This is the key, I think, to some of these early ruptures in tubal pregnancy. For instance an ovum (using this term to include an embryo and its membranes) the size of a walnut is suddenly enlarged to the size of an orange by hæmorrhage into its membranes. When lodged in the uterus this event causes sufficient disturbance to bring about expulsion of the ovum, accompanied by free bleeding: in the Fallopian tube this accident produces rupture, with discharge of the ovum into the peritoneal cavity, accompanied by profuse hæmorrhage; sometimes the extravasation takes place into the broad ligament, but in early cases this appears to be uncommon.

I do not base this opinion on one case. A few weeks ago I made a report on a similar specimen, and as the details

FIG. 1.—The distended Fallopian tube showing the situation of the rupture; *o*, ovary with corpus luteum.

FIG. 2.

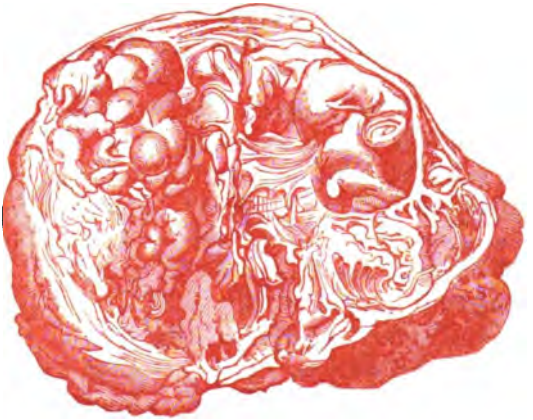

FIG. 3.

FIG. 2.—Apoplectic ovum from the Fallopian tube, represented in Fig. 1.
FIG. 3.—Distorted head of the fœtus. About seventh or eighth week of gestation.

of the case will probably be published shortly, further remarks upon it must not come from me.

Specimens of intra-peritoneal hæmatocele, as they are called, have been recorded and shown at societies, as examples of ruptured tubal pregnancies, but no embryo or membranes were found. I am strongly of opinion that no case should be regarded as due to ruptured tubal pregnancy unless membranes, or fœtus, or both, are forthcoming, however suggestive the clinical evidence.

The most noteworthy clinical facts in the case of Emma B— were these :

1. She had not been pregnant previously, although married seventeen years, yet the first pregnancy was tubal.
2. The rupture, though intra-peritoneal and accompanied by profuse bleeding, was not fatal.
3. The absence of the conspicuous signs of pregnancy such as enlarged breasts and vomiting.
4. This is, I believe, the first example in which an apoplectic ovum has been recorded as occurring in the Fallopian tube.

Note.—Shortly after this case was communicated to the Society the track of the drainage-tube reopened and discharged pus for a few weeks, until the three silk ligatures with which the pedicle was tied came away; it then closed, and has given rise to no further trouble.

May 23rd.—I saw Mrs. B— to-day, and found her in the best of health, and able to attend to household duties as formerly.

Addendum.—Since this paper was written and placed in the hands of the secretary I have, with the aid of the light it appears to furnish, re-examined a specimen of hæmatocele which has, I regret to say, been wrongly interpreted. As it admirably supports the contention of this paper I will briefly describe it.

Alice H—, æt. 25, came under the care of my colleague Dr. W. Duncan in August, 1886. She was married, had three children, the youngest being, at the time of her admission, two years old. Since the last confinement the

patient had suffered from pelvic pain and painful menorrhagia. Vaginal examination revealed an elastic swelling the size of a Tangerine orange to the left of the uterus. The swelling was regarded as a dilated Fallopian tube. In September, 1886, after consulting with my colleagues, I opened the abdomen and removed the uterine appendages. The left ovary was adherent to a fold of omentum which contained coagulated blood, and constituted the swelling which could be felt by the vagina. The ovaries were cystic, and the hæmorrhage was attributed to rupture of one of the enlarged follicles. The patient made an admirable recovery, and I preserved the hæmatocele as a pathological curiosity. My interest in the matter induced me to re-examine the specimen, with the following result:

One inch from the abdominal ostium of the Fallopian tube there is a rupture exposing for some distance the mucous membrane of the tube. Close beside this, embedded in laminated clot, is an apoplectic ovum of the bigness of a chestnut, and a few delicate fringes project from it. These, when examined microscopically, show the dendritic arrangement of the villi of the chorion. The hæmatocele in this case was not due to the rupture of a follicle, but to a ruptured tubal pregnancy of very early date (probably fourth or fifth week). It is the smallest apoplectic ovum I have as yet examined. From the history it is impossible to decide the date of the accident, but the hæmatocele had been noticed for many weeks previously to the operation, and there was no evidence of peritonitis beyond the few adhesions between the ovary, tube, and the omentum which immediately encysted the blood-clot.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 18.)

A CASE OF CHOLECYSTENTEROSTOMY.

BY

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Received September 21st—Read November 26th, 1889.

For the notes from which the history of this case has been abstracted I am indebted to my house surgeon, Mr. F. Hudson.

Mrs. V. B—, æt. 42, was admitted into the Leeds General Infirmary January 9th, 1888, on account of a tumour on the right side of the abdomen, which was accompanied by symptoms of acute peritonitis. She had had abdominal section performed a year previously by me for pelvic disease of several years' standing, which had produced confirmed invalidism, and after the removal of the cause, a right pyosalpinx, she had been able to resume her work, and for several months had enjoyed excellent health, and been able to perform very arduous duties as a general servant in a large family, her menstrual functions being regular and painless. Three weeks before admission she began to experience pain in the right side of the abdomen without apparent cause; she then noticed a small rounded swelling, tender on pressure, in the right hypochondriac region; there was no jaundice, and the bowels

were regular. The swelling and pain increased, and compelled her to give up her work. She was seen by her medical man, Mr. Loe, three days before admission, when there was very marked tenderness over the swelling, which seemed to be decidedly increasing. Her general condition became rapidly worse, and nothing could be retained on the stomach, the vomit being of a dark brown colour. She suffered from great pain and intense thirst, the abdomen being tympanitic and the pulse frequent and weak.

On admission to the infirmary on January 9th the patient had an anxious expression, and lay on her back with the knees drawn up. She complained of great pain in the right side of the abdomen, markedly increased by pressure, deep respiration, or turning on her side. There was a distinct sense of resistance in the right hypochondriac and iliac regions, with dulness on percussion; but on account of the extreme tenderness, palpation was rendered difficult. Respirations 30, pulse 130.

The fæces had a normal colour. The urine was normal except that it gave Gmelin's reaction for bile-pigment.

On January 13th, 1888, she became jaundiced. It now became evident that she would soon die unless relieved by operation, and on January 14th abdominal section was performed through the upper part of the right linea semilunaris, exposing a large cyst with thickened walls, which yielded by aspiration eight ounces of fœtid pus. After the gall-bladder had been emptied it was incised, and then explored, sponges having been previously packed round it. The finger was passed along the peritoneal surface of the cystic duct as far as possible, and beyond this, but inside the duct, was passed a long metal probe, this exploring as far as the junction of the cystic with the hepatic duct; the finger was also passed along the outside of the common duct as far as the duodenum, but no gall-stone or other obstruction could be felt. Around the common duct, as well as over the cystic duct, plastic lymph had been thrown out; and this probably explains the subsequent course of events.

The gall-bladder was stitched to the skin, and a drainage-tube inserted, the remainder of the wound being closed by silk sutures passed through all the layers of the abdominal wall, including the peritoneum.

For the first twenty-four hours the discharge remained clear, colourless, and mucoid ; in the second twenty-four hours it became slightly tinged with bile, and on the third day the discharge appeared to be pure bile. On the fourth day a smaller drainage-tube was inserted, and the stitches were removed on the seventh. The jaundice had quite disappeared forty-eight hours after the operation. The patient made an uninterrupted recovery with the exception of having a biliary fistula, through which apparently the whole of the bile was discharged ; for both the fæces and the urine showed no trace of biliary matter, either by inspection or on chemical examination.

During the fifteen months subsequent to the operation the patient's digestion was unimpaired unless she took too much fatty matter, and then she became sickly and lost her appetite, and rather more fat than normal was passed in the motions ; the bowels were quite regular without the use of aperients, and the odour was in no wise different from that of healthy fæces. Repeated measurements were made of the whole of the bile discharged during twenty-four hours, and a careful analysis of the bile thus collected was made.

The details of these and other observations will be considered in a separate paper, and may, I think, have an important bearing on the physiology of the bile, as may also observations made on the action of certain drugs on the biliary secretion have a bearing on biliary therapeutics.[1]

Now, although the patient was in good health, her condition was a very miserable one, since no apparatus could be made to fit sufficiently accurately to catch the whole of the bile, except when she was in bed. When

[1] Paper read before Royal Society (London), April 24th, 1890, and published in the 'Proceedings' of the Royal Society for 1890.

out of bed she had to catch the overflowing bile in absorbent cotton, which was retained in position by means of a bandage, thus necessitating her frequently changing her dressings and clothes. On one occasion, when she was unable to change the wool, the wearing of her bile-saturated garments gave her a severe chill, which resulted in an attack of pelvic cellulitis.

She was so miserable at the prospect of having to go through life with her fistula, that when I mentioned to her the possibility of again turning the bile into the bowel, she said she would risk anything to be rid of her trouble.

I asked my colleagues to see her with me, and they agreed that cholecystenterostomy was perfectly justifiable if its risks were fully explained to the patient.

Her consent was at once granted, and on March 2nd, 1889, I opened the abdomen in the right linea semilunaris through the old scar, in the centre of which was the fistula, prolonging the opening two inches beyond the lower end of the cicatrix. The gall-bladder was detached from the parietes, and found to be much contracted and thickened. There was so much matting of the viscera that it was found impracticable to bring up and fix the duodenum or jejunum to the gall-bladder as at first intended; hence the hepatic flexure of the colon, lying near, was raised and encircled by an elastic ligature, after its contents had been squeezed upwards and downwards. Convenient spots having been selected on the gall-bladder and colon, a circle the size of a florin was marked by a scalpel on each viscus. Along these lines, sutures of fine chromicised catgut were passed, about an eighth of an inch apart, by means of curved sewing-needles, but these were not tightened until openings a third of an inch in diameter had been made in the centre of the circles, quite through all the coats of the two viscera concerned, and the cut edges of the mucous membrane of the colon had been sutured by a number of interrupted stitches of fine catgut to the edge of the mucous membrane of the gall-bladder. The closed blades of a pair of Spencer Wells'

pressure forceps were passed through the opening from the gall-bladder into the bowel, in order to see that it was thoroughly patent after the ligatures had been tightened. The outer row of ligatures, only involving the serous and muscular coats, were tied and cut off short.

The refreshed edges of the old fistula were then brought together by means of a continuous catgut suture, the serous surface being tucked in and a number of Lembert's sutures being further applied over the line of union.

The elastic ligature was removed from the bowel, and the circulation became immediately re-established. The sponges which had been packed below and around the colon and gall-bladder had prevented soiling of the peritoneum.

A glass drainage-tube was placed in the right kidney pouch, and brought out at the lower end of the wound in order to guard against any accident of sutures giving way.

Lastly, silk sutures were employed to bring together the parietal incision in the usual manner. The patient had a little pain, but no sickness or distention.

On the night of March 3rd a tinge of bile appeared on the dressings, showing that the over-tense sutures on the outer surface of the gall-bladder had given way, but, thanks to the drainage-tube, without any dangerous result.

On the following day the bile came freely through the drainage-tube, and on March 5th fæcal matter made its appearance mixed with bile, after which, up to the 18th, fæces and bile continued to be discharged, and then bile alone, the wound granulating and ultimately completely closing on May 6th, when the motions were noticed to have fully regained their normal colour.

The patient, who was sent to a Convalescent Home, rapidly gained strength and weight, and reported herself in July as in perfect health. When she left the infirmary she weighed 8 st. 4½ lbs., and in July her weight was 9 st. 6½ lbs.

She was shown to the members of the British Medical

Association in Leeds in August, and then said that she had never been in better health.

During the time the fistula was open the menstrual functions were in abeyance. After its closure the menses returned, and have continued to recur regularly.

While the bile was being discharged externally Mrs. B— had a dislike to fat, to meat, and to sweet food, and a craving for acids such as lemons and pickles.

The operation of cholecystenterostomy was first performed by Winiwarter, who suggested its application in cases of irremediable obstruction in the common bile-duct. It has since been performed by Monastyrki, Kappeler, Socin, and Bardenheuer, but hitherto it has not been done in England. I think it has never previously been performed for biliary fistula.

The fact of the patient having within the space of four years undergone three abdominal sections is, perhaps, almost unique. Her first operation, the removal of a pyosalpinx, restored her to health and comfort after several years of distress and incapacity, besides relieving her from the constant danger of suppurative peritonitis. The second operation, cholecystotomy, for empyema of the gall-bladder, undertaken when she was apparently dying of peritonitis, undoubtedly saved her life. The third operation, cholecystenterostomy, performed for a condition which rendered her life wretched, has restored her to a condition of absolutely perfect health, for she is now strong, well, and healthy in every respect.

Numerous interesting questions arise in the case.

1st. On the benefit derived from removing diseased uterine appendages; and in this case only the diseased one was removed, the apparently sound one being retained, the menstrual functions being afterwards continued regularly and painlessly, except during the time the biliary fistula was open, when there was amenorrhœa.

2nd. On the advisability of operating during acute peritonitis in order to find out the cause, and, if possible, remove it.

3rd. On the treatment of distended gall-bladder by cholecystotomy, and not by cholecystectomy.

4th. On the cause of empyema of the gall-bladder.

5th. On the frequency or otherwise of fistula after cholecystotomy.

6th. On the apparent harmlessness to the system of the loss of the whole of the bile over so long a period as fifteen months, making it appear as if the bile were simply an excretion.

7th. The physiological experiment to which the patient voluntarily submitted herself, which will be discussed in another paper.

I would draw attention to some of the details in the operation described.

The cause of the fistula was apparently a cicatricial contraction of the duct. It was, therefore, hopeless to attempt to secure a return of the bile to the intestine by the ordinary channel, and on opening the abdomen it was found impossible, on account of the old adhesions, to stitch the gall-bladder to the small intestine in the region of the duodenum. It was therefore sutured to the colon. Instead of using the ordinary intestinal clamps, or passing ligatures through the mesentery and around the bowel, a loop of colon, after its contents had been squeezed out, was simply drawn up and secured at its base by an ordinary piece of elastic drainage-tube, which was fixed by a pair of pressure forceps. This tourniquet both prevented any escape of gas or fæcal matter, and rendered the intestine almost bloodless—in fact, it simplified the operation very considerably, and was applied in a few seconds; hence in future I shall never think of using any other intestinal clamp.

There is nothing calling for mention in the mode of application of the sutures, which were applied after the Czerny-Lembert method.

The loss of tissue from the outer surface of the gall-bladder, where it had been stitched to the skin for so long a period, rendered it necessary to apply more tension than

was desirable in order to secure exact apposition of serous surfaces. I therefore thought it wise to insert a glass drainage-tube into the right kidney pouch in case of the escape of any bile or fæces. This precaution prevented a catastrophe when the tense sutures gave way.

As I assumed that the escape came from the outer surface of the gall-bladder, and not from its junction with the intestine, I felt confident that the fistula would ultimately close by granulation, and that then the bile would be able to flow through the new channel. I am glad to say that this prognosis was justified by the course of events.

I must apologise for giving the history at some length, but I hope the interesting questions raised by this somewhat unique case may afford a sufficient excuse.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 28.)

ON

BLOOD TUMOURS (ANGEIOMATA AND ANGEIOSARCOMATA) OF BONE.

BY

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Blood tumours of bone are of great rarity, but nevertheless of considerable interest and importance, as their nature is very little understood, their diagnosis very difficult, and their treatment consequently not directed upon any definite lines.

The disease has received various names from different authors, having been called osteo-aneurism, capillary aneurism, hæmatoma of bone, sarcoma of bone, &c., these names expressing the different views which have been held with regard to its nature.

Its almost exclusive seat is the cancellous ends of the long bones, especially the head of the tibia and the condyles of the femur. On examination the tumour is found to be composed of a cyst-wall and contents. The cyst-wall is usually formed by a thin expanded shell of bone covered by thickened periosteum, to which the surrounding tissues are more than usually adherent. On opening the tumour it is found to contain little else but blood, partly fluid, partly

coagulated. Sometimes a thin layer of tissue is found lining the interior of the cyst-wall, and sometimes the bony shell is quite destitute of any lining. In some specimens the bone is so destroyed that there is complete loss of continuity between the portion of bone above and below the disease (see Fig. 1). In the last edition of one of the standard text-books of pathology[1] I read that these tumours "are now known to be in the majority of cases soft round-celled or spindle-celled sarcomata. . . . . They are exceedingly malignant, and hence the recognition of their sarcomatous nature is all-important." That this statement is not true of all cases is clearly proved by the following case which I had the opportunity of observing very carefully.

Lilian C—, æt. 4, was admitted into St. Bartholomew's Hospital on May 26th, 1886, under the care of Mr. Langton, to whom I am indebted for permission to publish the notes. The only history the mother could give was that ten months previously the child had fallen and hurt her leg. Since then she had noticed that her gait had altered somewhat, and that her left leg was gradually swelling just below the knee.

On admission her general health seemed excellent, and there was no evidence of any constitutional disease: the left leg was swollen below the knee, the circumference of the limb opposite the tubercle of the tibia being an inch and a half greater than that of the other limb. The swelling seemed to involve the upper three or four inches of the tibia and to expand it, for on pressure egg-shell crackling could be easily detected. There was complete absence of pain, tenderness, redness, and œdema; the knee-joint was natural, and there was no glandular enlargement to be discerned anywhere. The thigh was slightly wasted, probably from disuse. It will be gathered from the above account that the cause of the swelling was something inside the head of the tibia expanding it; that was sufficiently evident, but the nature of the "something

[1] Green's 'Pathology,' 1889.

inside" could only be guessed at. Thinking that the case was probably one of myeloid sarcoma, it was resolved to make an exploratory incision, and deal with the case according to the conditions found. This was done on June 14th, 1886, and it was discovered that the upper end of the tibia contained a cavity capable of holding about two ounces of fluid.

The wall of the cavity was composed of bone destitute of any lining, and in front, where the opening had been made, was not more than one tenth of an inch thick. Very little bone seemed to intervene between the cavity and the knee-joint. The contents consisted of a dark red fluid, looking like altered blood and serum, and a very little solid matter, which proved on microscopic examination to be blood-clot. The cavity was stuffed with oiled lint. In a few days it became lined by granulation tissue, and began to contract.

On August 18th the child developed scarlet fever, which necessitated her removal from the hospital; the cavity, however, continued to contract slowly, and by March, 1887, was completely obliterated, a healthy scar remaining in the site of the operation wound.

The patient is now perfectly well. There is a healthy scar in the site of the operation wound, and the bone seems quite consolidated. All the measurements of the two limbs are so nearly equal that no difference can be detected.

This case presented features so peculiar, and so unlike everything I had hitherto believed about such tumours, that I was induced to peruse the literature of the subject to see if I could discover anything like it. Although I could find no record of a similar case, yet I found accounts of others which I think throw very great light upon the nature of these tumours. I propose, therefore, to give short notes of those cases I have been able to find which have most bearing upon the subject.

The most malignant type of blood tumour of bone is nothing more or less than an endosteal sarcoma, in which

blood-vessels have burst and caused extravasation of blood. In this variety there still remains enough solid new growth to be at once recognised, even by the unaided eye, and on microscopic examination the nature of the tumour is sufficiently evident.

But sometimes the amount of sarcoma tissue is so remarkably small that it may easily escape detection. As an excellent example of this, I might quote the following case recorded by Max Oberst.[1]

A man twenty-one years old had noticed a swelling upon the inner side of his knee for three months. When first seen by Oberst the tumour was as large as two fists, and was fluctuating in some places. An incision was made into it and blood escaped, partly fluid and partly coagulated. The finger introduced through the aperture impinged upon the internal condyle of the femur, greatly destroyed. The limb was immediately amputated. On examining the limb the sac of the tumour was composed partly of the cancellous tissue of the lower end of the femur, and partly of a thin shell of bone greatly expanded and covered by thickened periosteum. The cyst-wall was lined inside by a thin layer of tissue, most marked in the interior of the femur. On microscopic examination this was found to be sarcoma tissue rich in large round-cells. There were a few giant-cells containing from five to fifteen nuclei. Only a very few layers of cells next to the periphery of the sac were intact; all the others were more or less disturbed and separated by effusion of blood.

The patient died subsequently with metastatic deposits of soft and vascular myeloid sarcoma, containing true ossifications in the periphery, and having a tendency to apoplexy.

Here, then, is a case presenting all the clinical characters of a sarcoma, but being peculiar in that the tendency to effusion of blood was far greater than the power of the tissue itself to grow, and hence the naked-eye appearances of the tumour on dissection.

[1] 'Deutsche Zeitschrift für Chirurgie,' Band xiv, 1881.

The following is a case in which the progress of the disease was arrested for seven years by tying the main artery of the limb. I have made the following abstract from Breschet,[1] who quotes the case from the practice of M. Dupuytren in the Hôtel Dieu.

Clement Nicholas R—, æt. 32, suffered from a pulsatile tumour expanding the upper end of the right tibia. He had noticed it for a year before his admission to the hospital on February 9th, 1819. Dupuytren regarded it as a case of osteo-aneurism, and tied the femoral artery on March 16th. The next day the tumour diminished in size, and on the sixth day the pulsations ceased. The patient left the hospital on April 30th, the "aneurism" having disappeared, leaving only a little tumefaction in the site formerly occupied by it. A long time afterwards the tumour grew again, and assumed a considerable size. On August 1st, 1826 (seven years after the operation), he again presented himself at the hospital, the tumour having attained such dimensions that the leg measured thirty-two inches in circumference. Dupuytren amputated above the knee, and the patient made a good recovery. The specimen was examined by Breschet. The limb was enormous owing to the extraordinary development of the upper end of the tibia, the condyles of which were expanded and divided by compartments into numerous cells like a pomegranate; the walls of the cavity were lined with a vascular network distended by injection, which had been forced into the arteries of the limb. Some of the cells contained a yellowish-black substance, others contained strata of coagulated blood. The cartilages, almost intact, were loosened from the osseous surfaces, and moveable in the middle of the disease.

The following case related by Roux[2] is even more remarkable.

A man, æt. 25, suffered from a pulsatile tumour ex-

[1] 'Repertoire Générale d'Anatomie et Physiologie Pathologique, et de Clinique Chirurgicale,' tome ii, Paris, 1826.

[2] 'Quarante années de pratique chirurgicale,' tome ii, p. 456.

panding the upper end of the tibia. After ligature of the femoral artery the tumour disappeared, and the bone returned to its normal condition. The patient was seen twenty years afterwards, and was then in good health. In this case it would seem that the inherent vitality of the new growth was so slight, that after a certain time it became arrested either by pressure of the extravasation of blood, or by arterial starvation following the ligation of the femoral artery, or both.

The following case related by Dr. Lagout d'Aigueperse[1] confirms the preceding case, which if it stood alone might be open to doubt.

The "aneurism" was situated in the upper end of the tibia. It diminished greatly in size, and its pulsation ceased after ligature of the femoral artery, but the bone did not return to its natural condition. It was in the same state eight years afterwards.

I will now quote a case in which the tendency to progress was so slight that it was arrested without any operative interference.

Dr. McDonnell,[2] in reading a paper on pulsating tumours of bone before the Royal Academy of Medicine in Ireland, detailed the case of a lady who had been sent to him by Mr. Erichsen nearly five years previously. She then suffered from a pulsating tumour over the upper part of the fibula, which he and Mr. Erichsen agreed in regarding as probably a hæmatoid sarcoma of bone. It continued for some time to increase in size. Operation was deferred on account of the lady's pregnancy, but she was directed to wear an elastic stocking. She suffered after delivery from phlegmasia of the other limb, which caused her to remain in bed for nearly six months—still, however, wearing the elastic stocking. When she came again under Dr. McDonnell's care some time after her parturition the tumour was found to have disappeared.

In the absence of any pathological examination of the

[1] 'Bulletin de la Société de Chirurg.,' t. ix, p. 258, 1858-9.

[2] 'Lancet,' Dec., 1888, p. 1130.

tumour it is impossible to be absolutely sure of the nature of the case; yet, taking into consideration the facts of the case, and bearing in mind that it was under the care of excellent observers, one can, I think, only infer that it was an innocent blood-cyst of bone, in which the newly formed tissue had such little power of growth that it was arrested by the pressure of an elastic stocking.

Here, then, we have a series of cases of blood tumour of bone presenting very varying clinical characters. Arranging them in order of malignancy, they may be briefly recapitulated thus:

1. Max Oberst's case. A highly malignant endosteal sarcoma.

2. Dupuytren's case. Arrested for seven years by tying the main artery of the limb.

3. Roux's case. Permanently cured by tying the main artery.

4. Lagout's case. Similar to the preceding.

5. The case recorded by myself, in which the growth was arrested by simple incision.

6. McDonnell's case. Cured by the pressure of an elastic stocking.

We must now turn to the microscopical characters of these tumours in order further to elucidate their nature.

In Oberst's case the microscope showed that the tumour was a myeloid sarcoma in which a large number of blood-vessels with very weak walls had burst, and caused extensive extravasation of blood, which had, so to speak, swamped and destroyed most of the solid tissue. It is to be regretted that the other cases in which the specimens were examined occurred before the days in which myeloid tumours had been recognised.

The piece of bone removed from the wall of the cyst in the case of Lilian C— was examined microscopically by my friend Mr. Bowlby, and by him and other authorities pronounced to contain myeloid sarcoma tissue. The section presents different appearances in different places. In some parts trabeculæ of bone are seen being eroded by small round

nucleated cells. In other places large giant-cells containing many nuclei are clearly seen, whilst the bulk of the tissue appears to consist of spindle-shaped cells very much like those of spindle-celled sarcoma. The blood-vessels of the tumour, however, are the most interesting and important. Many of them appear to be only spaces in the tissue with no wall of any sort, and from them red blood-corpuscles may be seen exuding into the surrounding tissues and crowding out the cells of the tissue itself (Fig. 3). Others have a very thin wall, still allowing considerable exudation. Others, chiefly the smallest, have a wall of considerable thickness, and containing many large nuclei crowded together. These blood-vessels are so numerous and large that one is at once led to inquire why they are present in such large numbers. Surely not because the tissue requires a great deal of nourishment, for we see plenty of examples of other tumours growing much more rapidly, and yet being much more poorly supplied with blood-vessels. I am, therefore, forced to conclude that the blood-vessels are an essential part of the tumour. But then it may be asked, how is it that these blood-vessels are mixed up with other structures which are evidently in many cases, at any rate, decidedly sarcomatous, both in microscopical appearance and in clinical deportment? I think the answer to this question is that these blood-vessels grow mainly, if not entirely, from the giant-cells of the bone-marrow, and may present every degree of developmental perfection, from the most imperfectly formed vessels incapable of containing blood up to the perfectly formed vessels of an ordinary angioma. This statement I am unable to prove, but there are many considerations tending to confirm my view.

Thus, firstly, Heitzmann[1] has described the formation of blood-vessels from the giant-cells found in the marrow of healthy cancellous bone. I am not aware that those observations have been confirmed by other observers, but

[1] Heitzmann, "Rück- und Neu-bildung v. Blutgef. im Knochen," 'Wien. med. Jahrb.,' 1873.

Dr. Klein tells me that although he has not actually observed the process himself, yet he thinks it highly probable on *a priori* grounds that it does occur; certainly, blood-vessels are developed from similar cells in other situations: thus in the area vasculosa of the chick large multinucleated cells may be seen becoming vacuolated and forming blood-vessels, and in the subcutaneous tissue of rats large multinucleated connective-tissue corpuscles may be observed to be undergoing the same changes. Although one cannot actually see blood-vessels developing from myeloid cells in the sections of the tumour I have described, yet there are appearances very suggestive of it, especially the capillary vessels with richly nucleated walls already described.

Secondly, these tumours only occur in those parts of bone where myeloid cells are found in health.

Thirdly, in several cases of blood tumour of bone, of which I have read the notes, great stress is laid upon the fact that the sac of the tumour contained a vast number of thin-walled vessels. Thus Breschet, in describing the tumour amputated by Dupuytren, says "the walls of the cavity are lined with a vascular network greatly developed. Over the membrane which lines some of the cells are seen vascular networks distended by the injection forced into the arteries." Scarpa,[1] in describing a blood tumour of the tibia for which he amputated the limb, says the "aneurismal sac was quite covered with arterial vessels of a much greater size than those of the proper arteries of the cellular substance and those of the periosteum. . . . . After cleaning thoroughly the inside of the aneurismal sac it was wonderful to see from how great a number of arterial orifices the wax injected into the popliteal artery had been effused into the cavity of the aneurism." Richet[2] observed the same appearances, and thought that these tumours were pure vascular tumours of bone, and denied that they contained any sarcomatous elements.

[1] 'Sull' Aneurisma,' fol., Patav., 1804.

[2] Richet, 'Archiv. Gén. de Med.,' 1864 and 1865.

Fourthly, pure angeiomata of bone are occasionally met with. Dr. Mapother[1] has recorded a case in which a blow upon the shin was followed by the development of a tumour, the size of a walnut, in the tibia; there was distensile pulsation, thrill, and bruit. He removed the cuticle by potassa fusa, and then applied a cautery. After an interval of ten days intense hæmorrhage occurred: in a few days a "nævoid matter" came away, leaving a granulated surface which rapidly healed. The patient was well sixteen years afterwards. A similar case also occurred to Dr. Bickersteth, of Liverpool. In these cases there was no blood tumour because the blood-vessels were sufficiently strong and fully developed to hold blood without bursting.

From the above facts and arguments I think it follows that not only do these blood tumours present different degrees of malignancy, but that they also differ in structure, some having very embryonic blood-vessels, others more fully developed ones; and it would appear that the more embryonic these vessels are, the more malignant are the clinical features of the tumour in which they occur; and conversely, the more the vessels approach to the type of fully developed structures, the less malignant the tumour. This notion is fully in accord with what is so well known about fibrous tumours, spindle-celled and round-celled sarcomata.

My conclusion, then, with regard to the nature of blood tumours of bone is that they are tumours of blood-vessels, some innocent and some malignant in nature. I would therefore suggest that they be called angeiomata and angeiosarcomata of bone.

Turning now from the pathological to the clinical aspect of these cases, we are still met by great difficulties. Owing to their great rarity they are seldom suspected, and hence usually unrecognised until they are subjected to surgical treatment. Their general symptoms have been sufficiently referred to in the preceding cases to need no further description. The most important thing

[1] 'Lancet,' Dec., 1888.

is to distinguish between innocent and malignant cases, for on this diagnosis must depend to a great extent our prognosis and treatment. On comparing the innocent and the malignant cases, the only difference discoverable between the two is that in the former the progress of the disease is slower than in the latter. As far as I can ascertain, there is no other difference which will help us to decide this very important point. Age seems to have no influence, nor do the characters of the tumour or its constitutional effect on the patient afford the least clue to its degree of malignancy.

How then should such cases be dealt with? The golden rule of cutting into the tumour before removal of the limb or affected bone should certainly be followed. If the tumour be found to contain a large quantity of solid sarcomatous-looking tissue there will be no chance for the patient except from very free removal, and this usually necessitates amputation. If, however, the contents of the tumour consist entirely of blood, or if only a very thin lining of tissue be found, I would suggest that the latter be scraped carefully away, and the cavity stuffed and allowed to granulate up. Of course there is a risk of dangerous hæmorrhage from the interior of the cyst-wall. In two cases, however, which I have seen opened (the one related above, and the other under the care of Mr. Cripps in St. Bartholomew's Hospital) no hæmorrhage occurred. Should it occur, one would not anticipate great difficulty in arresting it.

Should the future progress of the case indicate that we have to deal with a malignant growth we can still resort to amputation, and with scarcely less favorable prospect than if we had resorted to it in the first instance, while we have the satisfaction of having given the patient the chance of recovering with a sound limb.

Although ligature of the main artery of the limb has been practised in a few cases with success, I should not be disposed to try it, as, having regard to the great difficulty in distinguishing between an innocent and a malignant

tumour, we may be wasting valuable time in performing a useless operation which is not devoid of dangers peculiar to itself.

The other methods of treatment which have been used, such as injection with coagulating fluids, compression, &c., need only be mentioned to be condemned.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 42.)

DESCRIPTION OF PLATE I.

On Blood Tumours (Angeiomata and Angeiosarcomata) of Bone (EDMUND ROUGHTON, B.S.Lond., F.R.C.S.).

FIG. 1.—Blood-cyst of tibia (from the Museum of St. Bartholomew's Hospital).

FIG. 2.—Blood-cyst of lower end of femur.

a. Thin layer of sarcomatous tissue.

b. Cavity filled with blood.

(After Max Obërst.)

FIG. 3.—Blood-cyst of head of tibia (the case of L. C—).

Section of cyst-wall, showing blood-corpuscles exuding from thin-walled blood-vessels into surrounding tissues.

Fig 1

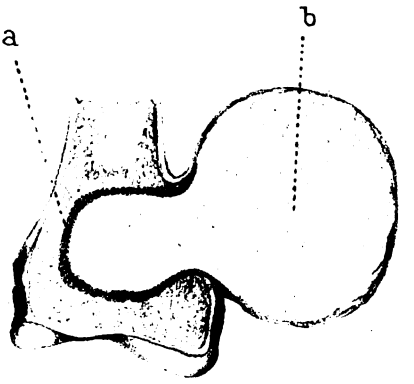

Fig 2.

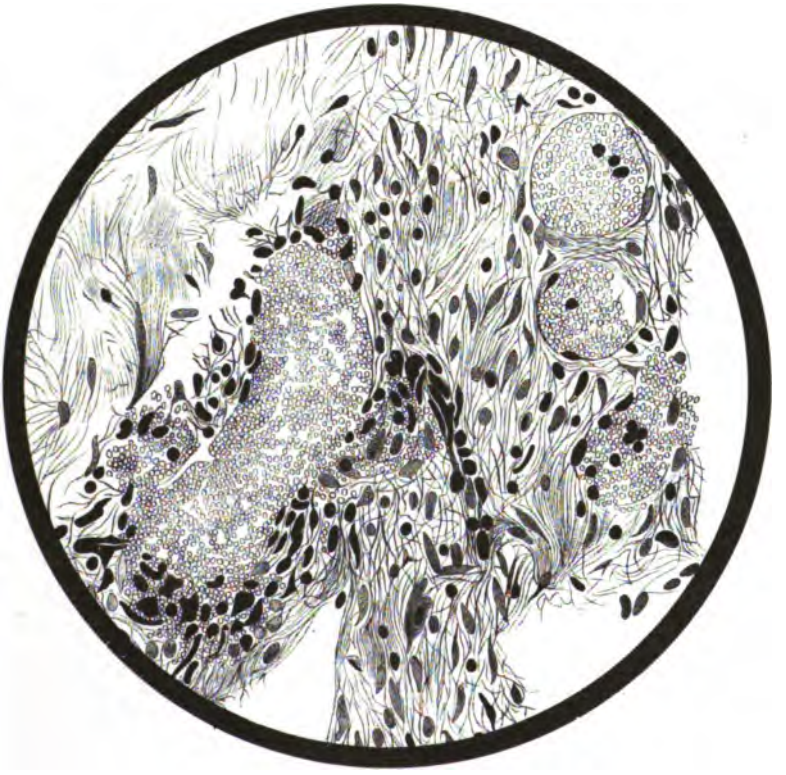

Fig 3

DANIELSSON & CO., LITHO.

SUCCESSFUL REMOVAL

OF THE

ENTIRE UPPER EXTREMITY FOR OSTEO-CHONDROMA.

BY

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On the recommendation of Dr. de Denne, of Cradley, a carter, æt. 40, was admitted into the General Hospital, Birmingham, January 16th, 1889, under my care, for a large tumour growing from the right humerus which ten years before had been first noticed below and external to the shoulder-joint. In spite of treatment the growth was steadily maintained, and the increase in size caused pain in the neck and forearm, but it was only during the last eighteen months that the surface became irregular and the growth perceptibly more rapid.

Three months before admission softened patches appeared at the lower part, and one week previously one such patch had given way and an oily fluid constantly drained from the opening. Up to the last the affected arm, with the aid of a sling, was used for driving, and there was no deterioration of the general health.

On admission.—A tumour twelve inches long, with a cir-

cumference of twenty-eight inches at its widest part and resting internally upon the thoracic wall, was found implicating the shaft of the humerus from a point immediately above the condyles, but the shoulder-joint was moveable.

Its surface was very irregular, and presented numerous bosses and depressions; most of the prominences were quite hard, but at the upper and outer aspect there was a fluctuating area, and at the lower part an ulcerated surface the size of a shilling. Numerous large dilated veins were seen running over the tumour.

The various internal organs of the body were apparently healthy.

January 19th.—There was a free oozing of venous blood from the ulcerated surface, but it was arrested by the application of an ice-bag, until the early morning of the 21st, when the hæmorrhage became so free that the ice failed to exert any beneficial influence, and the house surgeon was obliged to resort to the thermo-cautery. This reduced the bleeding to a slight ooze.

At 11 a.m. (January 21st) the following operation was performed:

Subperiosteal resection of the middle third of the clavicle by means of an incision carried along its shaft from the sterno-mastoid muscle to the acromion process of the scapula. The original intention was to tie the third part of the subclavian artery and vein with double ligatures, but, owing to free venous oozing resulting from division of some of the dilated veins, the first part of the axillary vessels were laid bare by carrying an incision from the middle of the clavicular one down the inner side of the tumour, and then backwards to the tip of the angle of the scapula, reflecting inwards the skin and dividing the pectoral muscles. The vessels were then divided, after the application of double silk ligatures, and the cords of the brachial plexus, together with the muscles forming the posterior axillary fold, severed. By turning the patient on the sound side and drawing the affected arm

across the chest, a posterior flap was formed by making an incision from the tip of the acromion process backwards to the angle of the scapula, and reflecting the skin to the posterior border of the bone. A division of the muscles attached to its upper and posterior borders permitted the removal of the scapula, the outer third of the clavicle, and the arm, *en masse*.

The exposed surfaces were irrigated with corrosive sublimate lotion, and the flaps brought together without tension by silver wire sutures. A dressing of iodoform and corrosive sublimate wool was applied, and the patient removed to bed.

By the following day (January 22nd) he had quite recovered from the effects of shock, and took light nourishment freely.

February 7th.—The upper portion of the wound had healed and the lower was granulating rapidly, but in the afternoon the patient, who had been sitting up in bed, experienced a shooting sensation in the wound, and immediately afterwards the dressings and adjacent bed-linen were found saturated with blood, the man becoming blanched and collapsed. The dressings were at once removed, but the wound presented no bleeding point, merely two small clots were visible in the position of the artery. An anæsthetic was administered, and the wound enlarged at its upper end by an incision an inch and a half long, parallel to the cicatrix of the former wound. It was then seen that the axillary artery was merely plugged with a small clot; this becoming dislodged a gush of blood took place. An aneurism needle, with a stout silk ligature, was then passed round the artery as it lay embedded in a mass of granulation tissue, and having been tied the ends of the silk ligature were left long.

On the 12th, the patient having completely rallied, as the pulsations of the artery were quite distinct down to the ligature, it was deemed prudent to reopen the wound made on the 7th, to saw off half an inch from the end of the clavicle, divide the deep cervical fascia and

secure the subclavian artery immediately behind the scalenus anticus. A silk ligature, with the ends left long, was employed.

The progress of the case from this date was uninterrupted.

On February 14th the ligature came away from the first part of the axillary artery in a very sloughy condition, but that from the subclavian did not separate until February 24th.

On March 6th the patient was able to get up, but for some time experienced a good deal of difficulty in balancing himself.

April 25th.—Dismissed.

Up to the present time (June, 1890) he has remained well and become considerably stouter.

The limb in its entirety was forwarded to Mr. Charles Cathcart, of Edinburgh, for the purpose of a cast being made according to his own method; and when that had been done to make a section of its growth. His report is as follows:

"The preparation consisted of the right upper limb, including the scapula and part of the clavicle, of a man. The weight of the preparation was 21 lbs.; that of the tumour about 18 lbs. The circumference of the tumour at its widest part was twenty-eight inches. The tumour was nodular in outline and of a firm elastic consistence, except at one or two places where it had softened and become cystic. In cutting the tumour the knife was greatly obstructed by calcareous nodules, and the saw and knife had to be used alternately.

"*Naked-eye appearance of fresh section.*—The tumour consisted essentially of nodules of clear cartilage, varying in size from that of a pea to that of a walnut. The smaller nodules were firm and transparent, like the substance of a crystalline lens. The larger ones were yellowish and somewhat opaque, and in some cases had softened in the centre. Round the nodules there was a delicate stroma of connective tissue containing blood-vessels.

"From the blood-vessels a deposit of calcareous salts had taken place in the periphery of the nodule, in some cases surrounding the nodule with a calcareous shell. Here and there, where several calcareous nodules in close proximity had formed a large mass, the structure of cancellated bone was distinctly visible, ossification having taken place.

"The upper and outer portions of the shaft of the humerus were thickened and sclerosed. The interior of the bone was apparently unaffected.

"Judging, therefore, from the specimen, the tumour would seem to have started from the periosteum at the upper end of the shaft of the humerus, towards the back and outer side; afterwards to have grown more and more away from the bone, pushing the soft parts before it."

Microscopically the tumour was found to be composed of hyaline cartilage, with no features of special interest.

Removal at one operation of the entire upper extremity and part of the clavicle, as a method of treatment in cases of neoplasmata situated in the region of the shoulder-joint, was first practised in 1838. From that date to 1863 four records only have been published. But since Mr. Syme's case in May of the last-named year there has been a steady increase in the number of records, and the operation is now a recognised procedure; a table of forty-four operations is herewith appended.

In 1882 M. Paul Berger, as the result of his observation and experimental investigations, was led to suggest that the operation should be systematically performed as follows:

1. Resection of the middle third of the clavicle. Professor Ollier in 1884 suggested that, as a safeguard against wounding the vessels, the resection should be made subperiosteally.[1] This he successfully performed, but Mr.

[1] Practically the subperiosteal resection is not to be recommended, as the periosteum left obscures the subclavius muscle, and has to be immediately divided.

Lund had previously in 1879 carried out this step in its entirety.

2. Double ligature of the third part of the subclavian artery and vein, and division of the vessels between the ligatures.

3. The formation of two oval skin flaps, an antero-inferior and a postero-superior, and removal of the entire limb with the remaining external portion of the clavicle *en masse*.

The advantages of following this order in the performance of the amputation are—

1. No hæmorrhage from the axillary artery and its branches can take place.

2. Entrance of air into the large vein is guarded against, and oozing from the vessels of the neoplasm is minimised.

3. An almost bloodless section of the pectoral muscles and the cords of the brachial plexus is permitted, whilst division of the posterior muscles, where the arterial supply has not been cut off, is reserved for the last step of the operation.

4. The flaps are readily approximated, and while there are no spaces for the pocketing of discharges, the facilities for drainage are excellent.

5. It permits a free and wide division of the various structures, this being of special importance when a malignant growth has to be dealt with.

6. The resulting stump readily admits of the application of an apparatus to hide the deformity.

Accepting this method as a basis, in the case here recorded two points are mainly brought into prominence :

1. The shock to the patient.

2. The securing of the vessels.

1. *The shock.*—During the operation this was marked when the large cords of the brachial plexus were severed. On removal from the table the effects were speedily rallied from. The general shock was apparently much less severe than that which has been noted in cases where amputation at the hip-joint has been undertaken for malignant tumours

connected with the femur. This observation confirms the experience of Berger and Bennett May.

The table appended shows that shock *per se* is not much to be dreaded.

2. *Securing the vessels.*—If from the nature of the tumour there be free venous oozing on incising the superficial tissues, or if there be structural displacement from invasion by the growth, it would seem to be better, after resection of the clavicle, to proceed at once to the formation of the upper portion of the antero-inferior flap, division of the two pectoral muscles, and fully to expose the axillary artery and vein. These can then be readily traced up to the scalenus anticus muscle, and the subclavian artery and vein secured.

Both Berger and May have found that in proceeding to direct double ligature of the vessels, after removal of the clavicle, considerable care was necessary to secure the vein in two ligatures; this would be obviated by tracing it from below upwards.

In this case the first part of the axillary artery was tied and divided above the acromio-thoracic branch, yet on the seventeenth day there was secondary hæmorrhage, and dissection showed patency of the main vessel. A ligature placed higher failed to diminish the pulsation of the artery, whilst tying the subclavian behind the scalenus anticus proved successful.

This experience seems to point to primary ligature of the subclavian as offering the best security against secondary hæmorrhage, for by this means the arterial supply is arrested as far as possible from the edges of the flap and sources of irritation.

It should, however, be noted that in the cases operated upon by Syme and Southam (Nos. 5 and 44) the axillary vessels were tied without any complication resulting.

Statistics.—The number of cases tabulated, in which the entire upper extremity has been removed for neoplasmata, is 43, and in one (No. 25) the operation was per-

formed for caries and osteo-myelitis, making a total of 44. For statistical purposes No. 28 may be excluded, as the operation not only consisted of removal of the scapula, but also of excision of the breast and portions of several ribs, thereby laying open the thoracic cavity.

From the immediate effects of the operation there were nine deaths. Of these, five were attributed to *shock*. Two (Nos. 9 and 24) succumbed the same day; two on the second day, No. 8 having fatty degeneration of several internal organs, and No. 18 having previously been a man of very intemperate habits; one (No. 41) died at the end of fifty-six hours, although the effects of the operation had apparently been recovered from.

Hæmorrhage caused two fatalities, No. 13 being due to loosening of the ligature securing the main vessels on the fifth day. No. 15 died the day after the operation.

Septicæmia (two), No. 7 on the fifth day; No. 17 on the sixth.

Fourteen cases *recovered* from the operation, to die at a later date with secondary deposits; one (No. 16) in three years; one (No. 12) in eighteen months; one (No. 32) in sixteen months; eleven (Nos. 1, 2, 19, 22, 30, 31, 35, 36, 37, 39, and 44) within the year.

One (No. 26) died of phthisis five months afterwards.

Ten cases may be counted as *cured*. No. 5 was living twenty-six years afterwards; No. 3, nine years; No. 21, six years; No. 20, five years. No. 23 lived three years, and death was not connected with the neoplasm. Nos. 6 and 11 were living two years after; Nos. 27, 42, eighteen months after. No. 25 was undertaken for caries from osteo-myelitis.

Uncertain, 10. For three (Nos. 14, 29, 38) no subsequent history is given; one (No. 40) was alive seventeen months after the operation; one (No. 43), thirteen months; two (Nos. 10, 34), twelve months; two (Nos. 4, 33) were living some months and three months respectively.

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Table of Cases.

No.	Operator.	Sex and age.	Disease.	Date of operation.	Method.	Result.	Reference.	Remarks.
1	MacClellan	M. 17	Large encephaloid growth of shoulder	1838	Removal of arm, scapula, and greater part of clavicle	Healed; death 6 months after, with secondary deposits	Rogers, Amer. Journ. Med. Sci., 1868	—
2	Twitchwell	—	Malignant growth	1838	—	Death in a few months from recurrence	New York Med. Journ., vol. viii, 1869	—
3	Mussey	Not given	Osteo-sarcoma	1845	Removal of scapula, arm, and external portion of clavicle	Healed; alive 9 years afterwards	Gross, System of Surgery, 1872	—
4	Whishaw	M. 8	Encephaloid growth of shoulder	1862	Removal of scapula, the outer third of clavicle, and the arm	Healed; alive some months afterwards	Lancet, vol. i, 1874, p. 819	—
5	Syme	M. 40	Fibro-chondroma of left humerus	May 7, 1863	Formation of semilunar flaps, clavicle divided at middle third; ligature of first part of axillary artery; removal of clavicle, scapula, and arm	Healed; alive in 1889	Monograph, Excision of Scapula, 1864. Communication from Mr. Annandale, present at operation (Nov., 1889)	Head of humerus resected Sept. 16, 1862. Tumour, size of walnut, removed from under cicatrix, Jan., 1863.

No.	Operator.	Sex and age.	Disease.	Date of operation.	Method.	Result.	Reference.	Remarks.
6	Fergusson, Sir Wm.	F. 19	Spindle-celled sarcoma of right scapula	Nov. 11, 1865	Clavicle divided just external to sterno-mastoid muscle; subclavian artery compressed against rib; removal of clavicle, scapula, and arm by semilunar flaps. Hæmorrhage slight	Healed by end of December; alive, and exhibited at King's College, Oct. 19, 1867	Med. Times and Gaz., Nov. 25, 1865	Infra-spinous portion of scapula removed with attached tumour, Jan. 14, 1865; recurrence in June.
7	Thiersch	M. 25	Enchondroma of left shoulder	Sept. 2, 1869	Disarticulation at shoulder-joint; clavicle, acromion process, and glenoid sawn off, and remainder of scapula removed	Death in 5 days, with symptoms of lung œdema	Adelmann, Archiv für klin. Chirurgie, Bd. xxxvii, 1888	Tumour previously removed in 1866. P.M.—Sero-purulent exudation in pleural cavity; enchondromatous masses round pulmonary vessels.
8	Fergusson, Sir Wm.	M. 40	Osteo-sarcoma, involving left scapula and shoulder-joint, 2 years' growth	Oct. 19, 1867	Clavicle divided at outer third; subclavian compressed; clavicle, scapula, and arm removed, vessels and nerves cut last. 6 oz. of blood lost	Death, Oct. 21, 1867	Lancet, Nov. 2, 1867	P.M.—Fatty degeneration of internal organs.
9	M'Leod, Kenneth	M. 2	Sarcoma (mixed-celled) extending from right elbow to infra-spinous fossa	June 30, 1869	Formation of flaps; ligature of axillary artery and vein under pectoral muscle; removal of part of clavicle, the scapula, and arm	Died same day	Edin. Med. Journ., Dec., 1869	Tumour, said to be congenital.
10	Hamilton	M.	Colloid tumour of scapula, 6 years' growth	Dec. 12, 1870	Digital compression of subclavian; large anterior flap; removal of scapula and arm	Healed by Feb., 1871	New York Med. Record, 1871–2	Excision of tumour in 1865; recurrence and removal in 1868; attempted removal in 1869 frustrated by hæmorrhage.

11	Barwell	M. 47	Osteo-sarcoma of scapula, involving head of humerus	Dec., 1870	Subclavian artery ligatured; disarticulation at shoulder-joint; outer third of clavicle and scapula removed separately	Healed; alive in 1872	Communicated by Mr. Barwell, Nov., 1889	—
12	Parise	M. 30	Osteo-sarcoma, double size of fist, involving head of right humerus, glenoid cavity, and adjacent structures	1873	Ligature of third part of subclavian artery and vein; removal of arm and scapula (except a strip of 2 centimetres at posterior border), with external portion of clavicle	Healed; death 18 months afterwards with secondary deposits	Langenhagen, Contribution à l'étude clinique des Tumeurs du Scapulum, Paris, 1883	—
13	Langenbeck, B. von	M. 17	Sarcoma of right shoulder, 15 months' growth	Dec. 19, 1873	Removal of part of clavicle, scapula, and arm	Death in 5 days from hæmorrhage	Veit, Extirpat. von Schulterblatt und Arm, Berlin, 1874	Hæmorrhage due to slipping of ligature applied to subclavian artery.
14	Esmarch, Fr.	M. 50	Myxo-sarcoma in right axilla, 5 months' growth	July 8, 1874	Ligature of subclavian artery and vein; removal of scapula and arm	Healed (?)	Adelmann, Archiv für klin. Chir., Bd. xxxvii, 1888	—
15	Macnamara, Charles	F. 24	Sarcoma (mixed-celled), size of infant's head, in region of right scapula, and passing under clavicle, 4 years' growth	Mar. 27, 1878	Removal of part of clavicle, entire scapula, and arm. Owing to position of neoplasm, the subclavian artery could not be tied until limb removed; severe hæmorrhage	Death, March 28, 1878	Lancet, May 11, 1878	Death attributed to loss of blood.
16	Lund, Edward	M. 20	Sarcoma (spindle-celled), extending from margin of left deltoid, filling the axilla and overlapping the scapula, 13 weeks' growth	Oct. 3, 1879	Resection of middle third of clavicle; formation of flaps; ligature of subclavian artery and vein; removal of scapula, outer third of clavicle, and arm; 3 oz. of blood lost	Healed; death from recurrence, Sept., 1882	Brit. Med. Journ., Oct. 16, 1880; T. Jones, Diseases of the Bones, 1887, p. 334	Exploratory incision of neoplasm, Sept. 26; recurrent growth was attached to three upper ribs, spread over apex of lung, and enveloped the aorta and inferior vena cava.

No.	Operator.	Sex and age.	Disease.	Date of operation.	Method.	Result.	Reference.	Remarks.
17	McGill	F. 58	Sarcoma, attached to axillary border and lower portion of right scapula	May, 1880	Formation of flaps; division of outer third of clavicle; ligature of subclavian artery and vein; removal of detached clavicle, scapula, and arm	Died on the 6th day	Brit. Med. Journ., Oct. 30, 1880	Flaps sloughed; death due to septicæmia.
18	Whitehead, Walter	M. 24	Sarcoma of right humerus, involving shoulder-joint, 8 months' growth	Jan. 26, 1881	Removal of outer third of clavicle; ligature of first part of axillary artery and vein; skin flaps formed; scapula and arm removed	Death, Jan. 27, 1881	Communicated by Mr. Whitehead, Nov., 1889	Patient was a barman, and very intemperate.
19	Després	M. 22	Recurrent osteo-sarcoma, involving scapula, skin, and axillary glands	June 19, 1882	Ligature of subclavian artery; formation of flaps; division of outer third of clavicle, and removal of it, with scapula and arm (entrance of air into subclavian vein)	Healed in 2 months; death in 7 months, with secondary lung deposits	Langenhagen (quoted at No. 12)	Six months before, part of infra-spinous fossa had been removed with primary growth.
20	Berger, Paul	M. 27	Osteo-chondroma of right arm and shoulder	Oct. 28, 1882	Resection of middle third of clavicle; double ligature of subclavian artery and vein (third part); removal of outer third of clavicle, scapula, and arm	Healed in 4 months	Monograph, L'amputation du Membre Supérieur dans la contiguité du Tronc, Paris, 1887	Right forearm amputated for tumour on dorsum of right hand when 12 years old.
21	Heath, Christopher	M. 16	Sarcoma (spindle-celled, ossifying) of upper end of right humerus implicating scapula	July 4, 1883	Disarticulation of shoulder; scapula and outer third of clavicle removed separately	Healed in 59 days; alive in 1889	Lancet, March 1, 1884 (communication from Mr. Heath, Nov., 1889)	Recurrence in February (7th month) in cicatrix; removed. Second recurrence in 35 months; removed.

22	Verneuil	M. 23	Sarcoma (round-celled) of upper end of right humerus, implicating scapula and muscles	Nov. 2, 1883	Division of the middle third of clavicle. Double ligature of subclavian artery and vein, with division; acromion sawn through at level of clavicle and the arm removed; then the remainder of scapula taken away; 7 oz. of blood lost	Left hospital on 17th day with wound, superficial	L'Union Médicale, Jan. 1, 1884	Recurrence of disease in dorsal vertebræ in May, 1884 (Berger).
23	Watson, P. Heron	M. 33	Enchondroma of right humerus, 18 months' growth	Dec. 17, 1883	Formation of oval skin flaps; division of clavicle at middle third, and ligature of subclavian vessels; removal of scapula, arm, and outer third of clavicle	Healed; died 3 years after; exact cause of death not ascertained	Edin. Med. Journ., Feb., 1884 (communication from Dr. Watson MacGillivray, Nov., 1889)	Enchondromata of fingers of both hands from early life; right middle finger amputated by Syme.
24	M'Leod, Kenneth	M. 20	Sarcoma (mixed-celled) of right humerus, 3 months' growth	July 28, 1883	Removal of portion of clavicle, scapula, and arm	Death same day (shock)	Indian Med. Gaz., Jan., 1884	P.M.—Recent infarcts in both lungs.
25	Bérenger-Féraud	M. 23	Caries of left scapula; osteo-myelitis of humerus; destruction of shoulder-joint	May 20, 1884	Digital compression of vessels; subperiosteal resection of scapula; removal of arm; clavicle not interfered with	Healed entirely, Jan. 22, 1885	Bull. de Thérap., Nos. 11 and 12, 1885	—
26	Czerny	M. 41	Sarcoma (spindle-celled)	July, 1884	Removal of clavicle, scapula, and arm	Healed in 24 days; death in Dec., 1884, due to pneumonia and laryngeal phthisis	Adelmann, Archiv für klin. Chir., Bd. xxxvii, 1888	Tumour previously removed in Dec., 1885.

No.	Operator.	Sex and age.	Disease.	Date of operation.	Method.	Result.	Reference.	Remarks.
27	Ollier	M. 50	Myxo-sarcoma, originating in right scapula, implicating the soft parts round the shoulder-joint	Oct. 29, 1884	Subperiosteal resection of middle third of clavicle; double ligature and division of subclavian vein and artery; removal of outer portion of clavicle with scapula and arm	Left hospital Dec. 4, 1884; alive in April, 1886 (Berger)	Lyon Méd., Feb. 1, 1885	The silk ligatures applied to vessels separated in May, 1885 (Berger).
28	Domenico, Morisani	F. 54	Carcinoma of right breast, thoracic wall, axillary lymphatics, and surrounding tissues; 16 or 17 years' growth	Mar. 15, 1885	Removal of clavicle at junction of middle and internal thirds; ligature of subclavian artery and vein; removal of scapula, arm, neoplasm, and surrounding soft parts, together with several inches of the second, third, and fourth ribs; right thoracic cavity opened	Death from shock five hours afterwards	Il Morgagni. Agosto, Ottobre, 1885 (quoted by Berger)	—
29	Bergmann	M. 28	Sarcoma (periosteal) of right humerus and scapula; a year's duration	Mar. 23, 1885	Primary ligature of subclavian artery and vein; removal of scapula and arm	Healed in 7 weeks	Bramsfeld, Ueber enige Fälle von Schulterblatt Extirspat., Berlin, 1888	—
30	Bell, Joseph	M. 30	Sarcoma (round-celled) of scapula involving humerus	May, 1885	Removal of portion of clavicle, scapula, and arm; hæmorrhage restrained by "skewer" passed under clavicle and vessel and rubber cord looped round	Healed; recurrence; death in a year	Manual of Surg. Operations, 6th edit. (communication from Dr. Bell, May, 1890)	—

31	Maling, E. A.	M. 50	Sarcoma (mixed-celled) originating in left infra-spinous fossa, extending into axilla, and involving shoulder-joint	Jan. 22, 1886	Ligature of third part of subclavian artery; clavicle divided and taken away with scapula and arm	Healed by April 2, 1886; death with secondary deposits	Brit. Med. Journ., Dec. 11, 1886	—
32	Bergmann	M. 44	Sarcoma of left humerus and axillary glands; six months' growth	July 15, 1886	Primary ligature of subclavian artery and vein; removal of scapula and arm	Healed by Aug. 3; death with secondary deposits in lung and pleura, Nov., 1887	—	—
33	Poggi, Alfonso	M. 27	Fibro-sarcoma of right shoulder; a year's growth	Oct. 13, 1886	Removal of acromial half of clavicle; ligature of subclavian artery and vein; formation of skin flaps; removal of tumour with scapula and arm	Healed in 41 days; remained well 3 months afterwards	Adelmann, Archiv für klin. Chir., Bd. xxxvii, 1888	—
34	Madelung	M. 42	Enchondroma of left humerus size of man's head	Dec. 9, 1886	Removal of most of clavicle, scapula, and arm	Healed in 47 days; alive 1 year afterwards	Zeits. für Chirur., Bd. xxvii, 1888	Trendelenburg had previously in 1880 removed tumour from humerus.
35	Obalinski	F. 22	Sarcoma	Feb. 6, 1887	Primary ligature of subclavian artery and vein; removal of scapula and arm	Healed; death in 3½ months with recurrence in pleura	Wiener med. Presse, No. 19, 1887	—
36	Reyher, Carl	M. 57	Sarcoma (spindle-celled) of left shoulder	Feb., 1887	Removal of clavicle, scapula, and arm	Healed; death in 11 months from recurrence in lungs	Adelmann, Archiv für klin. Chir., Bd. xxxvii, 1888	—

7

No.	Operator.	Sex and age.	Disease.	Date of operation.	Method.	Result.	Reference.	Remarks.
37	May, Bennett	F. 21	Sarcoma (mixed-celled, ossifying in places) growing from upper end of right humerus; 2 years' growth	June, 1887	Subperiosteal resection of middle third of clavicle; double ligature of subclavian artery and vein; removal of scapula, outer third of clavicle, arm, and infiltrated supraclavicular glands	Healed in 3 weeks; rapid recurrence in cervical glands; death	Ann. of Surg., December, 1888	—
38	Bergmann	F. 34	Sarcoma of left humerus	Jan. 24, 1888	Ligature of subclavian artery and vein; removal of part of clavicle, scapula, and arm	Healed	Archiv für klin. Chirur., Bd. xxxvii, 1888	—
39	May, Bennett	M. 17	Sarcoma (round-celled) growing from periosteum of upper end of right humerus	April, 1888	Subperiosteal resection of middle third of clavicle; double ligature of subclavian artery and vein; removal of scapula, outer third of clavicle, and arm	Healed in 1 mo.; recurrence; death within a year	Ann. of Surg., Dec., 1888	—
40	Parkes, C. T.	M. 18	Sarcoma (round-celled) of humerus, involving shoulder-joint	Dec. 11, 1888	Ligature of subclavian artery; removal of scapula and arm	Healed in 6 weeks; alive in May, 1890	Journ. Amer. Med. Assoc., Mar. 2, 1889 (communication from Dr. Parkes, May, 1890)	—
41	Parkes, C. T.	M. 37	Sarcoma (round-celled) of head of humerus, involving shoulder-joint	Jan. 8, 1889	Resection of middle third of clavicle; double ligature of subclavian artery and vein; removal of scapula, outer third of clavicle, and arm	Death in 56 hours from shock	Ditto	Exploratory incision into tumour made in Dec., 1888.

42	Chavasse	M. 40	Osteo-chondroma of right humerus, 10 years' growth	Jan. 21, 1889	Subperiosteal resection of middle third of clavicle; double ligature of first part of axillary artery and vein; removal of scapula, outer third of clavicle, and arm	Healed; alive in June, 1890	—	Profuse secondary hæmorrhage on 17th day; ligature of second part of subclavian artery.
43	Lewis, E. A.	M. 47	Sarcoma (myeloid ossifying) of head of left humerus	April 29, 1889	Ligature of subclavian artery; removal of scapula with half of clavicle and arm	Healed; no recurrence at date of publication	Annals of Surgery, Feb., 1890	—
44	Southam	F. 11	Sarcoma (round-celled) originating in subspinous fossa, involving axilla, and reaching as high as clavicle	Aug. 25, 1889	Formation and reflection of oval flaps; resection of outer half of clavicle; ligature of first part of axillary artery; removal of scapula, outer half of clavicle, and arm	Healed; left hospital Oct. 4; death in Jan., 1890, from recurrence	Brit. Med. Journ., Dec. 14, 1889 (communication from Mr. Southam, June, 1890)	—

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 48.)

DESCRIPTION OF PLATE II.

Successful Removal of the Entire Upper Extremity for Osteo-chondroma (THOMAS F. CHAVASSE, M.D., C.M.Edin.).

FIG. 1.—Half-length portrait of subject, showing tumour growing from right humerus.

FIG. 2.—Half-length portrait, showing subject after recovery.

Fig. 1.

Fig. 2.

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THE MECHANISM OF SUSPENSION

IN THE

TREATMENT OF LOCOMOTOR ATAXY.

BY

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THE treatment of locomotor ataxy by suspension has engaged so much attention of late, and has been advocated and attacked with so much energy on either side, that a serious effort to assign it a rational basis would have been at any time a welcome contribution to the discussion. It does not appear, however, that such an effort has been made hitherto; and this is the more strange because the dangers to which the proceeding is open are very generally admitted, while at the same time the benefit with which it is occasionally attended is supported by ample testimony.

Thus it happens that the matter is invested with a double interest, and from either point of view it is highly important to attain to a right understanding of it.

If, moreover, it could be shown that the danger of the operation is not inseparable from its admitted advantages, but rather that the latter can be better ensured by eliminating the element of risk, a practical conclusion of some

consequence will result. To establish such a conclusion is the object of this paper. It further pretends to show the methods of reasoning and observation by which the writer has been compelled to dissent from the views of others more competent to judge than he is, but perhaps in some cases without the same opportunities of testing the truth of their convictions.

It cannot be said that the suspension treatment is without its theory. Charcot in France, and de Watteville in England, to whom belongs the credit of its adoption in their respective countries, left the matter open. Charcot suggested that the effect might be due to a stretching of the spinal cord, or of the nerve-roots, but he thought it possible also that it might be caused by changes in the spinal blood-supply. A stretching of the cord, however, would seem to be the condition aimed at in practice. The origin of the treatment is known to everyone. The physician of Odessa who first resorted to it did so on the grounds of an apparent cure in the case of a patient who, in addition to tabes, had caries of the spine; and in that case no doubt the cord, or rather its membranes, were stretched in the process. There is no need to point out that for the purpose in hand the analogy fails, in an important particular, between those instances where caries of the vertebræ exists and those where it does not. Perhaps it is this coincidence which has biassed men's minds and made the assumption very general of a fact which is far from self-evident. That the assumption is general appears sufficiently from the reports of cases, where considerable elongation of the vertebral column is recorded, and improvement or disaster accounted for thereby. From those who reject the procedure little is heard of the formidable danger of dislocation, and a great deal of hidden menace from tampering with the cord. By its supporters the prospect of a cure is openly or tacitly referred to the same indefinite agency. The theory was boldly enunciated in England by Dr. Althaus. Writing in the 'Lancet' April 13th, 1889, he says: "Part of the

influence of suspension *by which the cord is efficiently stretched* is owing to the breaking down of adhesions due to chronic meningitis;" and again: "by the process of *stretching the spinal cord*, the overgrown and unduly hardened neuroglia may be loosened and broken down." This theory was endorsed by Prof. McCall Anderson; speaking at Glasgow in October, he referred to it as the best yet announced.

We need not pause to ask whether the symptoms of locomotor ataxy may be held to depend in any degree upon meningitis, or whether the most powerful stretching would be adequate to change the character of a sclerosis; but does the needful stretching occur, or is it not rather a *deus ex machinâ?* To answer this question it will be necessary to consider the evidence upon which the belief may be thought to rest. The writer knows of no other than certain measurements made during life. Some of these partake of the marvellous. Thus Dr. Bianchetti, of Padua, by suspending certain heavy men extended the vertebral column (by which, of course, the line of the spines is meant) as much as 4 centimetres (nearly $1\frac{5}{8}$ inches). At the same time, in three cases out of eight amaurosis was induced, and this leaven of mischief amidst much good was ascribed to an extension of the cord in some degree comparable to the above-mentioned elongation behind it.

The reputed fact and the inference are not in logical sequence, but we are concerned now with the statement. Taken for what it implies—an absolute elongation of the column to that extent—it is incredible. The vertebral column is not straight, but deflected in curves (Fig. 1), and the first effect of the force represented by the weight of the body is to straighten out these curves. Not till straightening has occurred will any considerable elongation of a convexity take place. Now the dorsal curve is much the largest, and its convexity is behind, where measurement has to be taken. To obtain so much as 4 centimetres, therefore, would be needed either a formidable extensibility

of the powerful ligaments which knit together the spines, laminæ, and transverse processes in the cervical region,

FIG. 1. To show the vertebral curves with the spinal canals exposed. (Drawn from the skeleton.)

or such a hiatus between the bodies of the dorsal vertebræ anteriorly as the imagination fails to supply.

Very little experience suffices to show the direction from which misconceptions of this kind arise. During life the vertebral column can be measured only along the summits of the spinous processes. How far an inference is warranted from such a measurement to the condition of the cord will be considered presently. The limitations of the process of measurement itself demand some notice now.

In flexion of the head the spines from the first to the seventh cervical are separated, so that a tape placed along their summits will measure upwards of eight inches. When the head is thrown back the spines and laminæ overlap, so that the same points are approached within three inches. Similarly in stooping the dorsal spines will spread over an area exceeding by three or four inches their extent when approximated by muscular action. Again, extension and depression of the occiput will cause a notable difference. It is always extremely difficult to ascertain differences due to muscular tension, and consequently to place the body in a position in this respect similar to that which it assumes when suspended. When an effort is made the decision must be in any event purely arbitrary. The difficulty described must have been appreciated by everyone who has conscientiously endeavoured to obtain the measurements required, and he will have satisfied himself that an error of more than two inches can be accounted for in this way. After much experience, I am convinced that comparative measurements of this kind taken on the living body are but little reliable. There is great difficulty in ascertaining the points chosen; and the time during which suspension is tolerated, at any rate where the patient is not supported from the axillæ, hardly suffices to make sure of them. In the statistics which I have prepared I have done what I could to obviate this defect. I have secured the assistance of skilled anatomists, considered with them every chance of fallacy, and controlled the results by others derived from observation on the dead body. Proceeding in this way I have come to the conclusion that in the living subject there is a decrease in the length

measured along the spines from the second cervical to the last lumbar vertebra of less than one third of an inch; that the portion of the column which is occupied by the dorsal curve is contracted by about half an inch, and that consequently the apparent lengthening takes place entirely in the neck. Apparent lengthening is said, because it is impossible to be sure that in the original estimate the proper state of muscular equilibrium has been maintained.

The patient is instructed to stand erect, with the chin and occiput inclined in a suitable manner. The observer then measures with a tape the entire length of the vertebral column, following the prominence of the spines. This is noted. The spine of the axis is then made out, and the distance from its lower border to that of the fifth lumbar is measured and written down. The same points are taken while the patient is suspended. The process is somewhat tedious, and in the last case is apt to be hurried, which itself adds an element of ambiguity. Some of the results obtained by me are appended. In no case were marks on the skin trusted to, but the bony point aimed at was determined by the finger on each occasion.

The results obtained in this way are so much at variance with others reported elsewhere that I feel called upon to point out the probable sources of error. Allusion has already been made to that which depends upon muscular action. Measurements of the neck, which is so much more freely movable than the back, are especially open to fallacy of this kind—so much so indeed that figures connected with it are but little trustworthy. On the living body my estimate, conjointly with that of others, has varied in successive experiments on the same person. Accordingly I attach but little importance to the result of observations on this part. I hope, however, to make it appear that variations in the length of the neck are of minor consequence, and I have endeavoured to attain securer data by experiments upon the dissected subject.

The objection indicated does not apply with equal force to careful measurements of the dorsal and lumbar curves,

but neither are these devoid of difficulty. Thus it is not always easy to determine the points chosen. In the statistics produced here the lower border of the spine of the first dorsal vertebra was invariably chosen as a fixed point, because that spine is the most prominent. But here, even in persons in whom the bony points stand out well, an error is very apt to arise. In such persons the observer has satisfied himself that his finger tip was on the extreme border, and when the patient was told to flex his back the true point proved to be a quarter of an inch lower down. When the body is suspended the point in question is most prominent. The difficulty then occurs in the preliminary measurement, and it can be surmounted with care. It depends upon the obliquity of the spinous processes. The posture assumed with the body erect is practically the same under all circumstances, so far as the dorsal curve is concerned. Consequently it is possible. though not easy, to secure absolute certainty in measuring that region. The same is true of the lumbar curve. But it appears that an error of a quarter of an inch may occur in determining one fixed point and as much in determining the other, not to speak of grosser mistakes, such as taking the first lumbar for the last dorsal spine, or the fourth for the fifth; and, since variations in the length of the dorsal and lumbar regions together seldom exceed half an inch, the need of accuracy will be appreciated. In order to supply this we have controlled our observations on the living body by dissections of the dead. The results are appended of only six cases chosen from amongst many, because they were judged to be most nearly correct, and because they were obtained with the assistance of others.

October 30th, 1889. *Regent's Park Hospital.*

CASES 1 and 2.—Dr. Cagney suspended by Mr. Roughton.

Posterior measurements :

		Suspended.		
	(*a*) Standing.	(*b*) Occip.—chin.	(*c*) Occip.—axillæ	
i. 2 C. to 4 L. (spines) ...	(22) 22⅜ in.	... 22½ in.	... 22 in.	
ii. 2 C. to 1 D. „ ...	3½	... 4½	... 4½	
iii. 1 D. to 4 L. „ ...	(18½) 18¼	... 17¾	... 17½	

Analysis.—Questionable decrease on the whole in (*b*) and (*c*). Absolute increase of about 1 in. in cervical region, similar diminution in dorsal. The dorsal region was still more shortened in (*c*), where the axillæ were supported.

CASE 3.—Mr. Roughton suspended by Dr. Cagney.

Posterior measurements :

	(*a*) Standing.	(*b*) Suspended from occip. and chin.
i. 2 C. to 4 L. (spines) ...	(22⅛) 21⅞ in.	... 21⅝ in.
ii. 2 C. to 1 D. „ ...	3⅜	... 3⅛
iii. 1 D. to 4 L. „ ...	18¼	... 18

Analysis.—Total diminution of ⅛ in. Increase of ¼ in. (?) in cervical, diminution of ¼ in. in dorso-lumbar curves.

Mr. Roughton fainted when taken down.

CASE 4.—Wm. B—, æt. 47. Tabes. Often suspended before. Reports having fainted first time. Has a considerable lateral curvature.

Posterior measurements :

	Standing.	Suspended (occip.—axill
i. 2 C. to 1 D. (spines) ...	3⅛ in.	... 3⅛ in.
ii. 1 D. to 4 L. „ ...	17⅛	... 17¼
iii. 2 C. to 4 L. „ ...	20¼	... 20⅜

Analysis.—Total elongation of ⅜ in. Increase of ⅜ in. (?) in cervical, ⅛ in. in lumbo-dorsal region.

This is the only instance met with in which the dorsal region was stretched, and it is remarkable that the patient had curvature of the spine.

November 5th.

Case 5.—Wm. D—, æt. 40, cook. Paræsthesia of trunk. First suspension.

Posterior measurements:

	(*a*) Standing before suspension.	(*b*) Suspended.
i. 2 C. to 1 D. (spines)	... 4½ in.	... 4½ in.
ii. 1 D. to 5 L. „	... 15¼	... 15½

Analysis.—Increase in cervical region of ⅜ in. (?), diminution in dorsal of ¼ in. Total increase of ⅛ in. (?).

Case 6.—Ch. H—, ataxic. Fourth suspension.

Posterior measurements.

	(*a*) Standing before suspension.	(*b*) Suspended.
i. 2 C. to 1 D. (spines)	... 4 in.	... 3½ in.
ii. 1 D. to 5 L. „	... 16½	... 16½ (?)

Analysis.—Diminution of ½ in. in cervical region and total diminution of ½ in. Dorsal measurements somewhat doubtful.

Reference has been made to the uncertainty of measurements of the neck during life. It is illustrated here by the alternative figures quoted in two cases. Both sets were thought to indicate the distance along the spines with the head as erect as possible. The difference is so great that no conclusions can be drawn from the figures, and they have been retained because they seemed the most nearly accurate that could be found, and also because they tally with others more reliable derived from the dead body. Read in connection with these, certain inferences may be based upon them.

The measurements of the dorsal and lumbar regions may be regarded as quite exact in all but one case.

The subjects of experiments were in some cases healthy, in others they suffered from spinal disease.

In every instance except one there was a *contraction* in the dorso-lumbar region, *not an elongation*. In this excep-

tional instance it was noted beforehand that the patient (a tabetic) had an extensive lateral curvature. The elongation in this case did not exceed $\frac{1}{8}$ in. In another case the dorso-lumbar measurement was invariable, $16\frac{1}{2}$ in. from the first dorsal to the fifth lumbar spine both before and during suspension. The greatest contraction occurred in the writer's own case when he was suspended, and the notes were made by his colleague, Mr. Edmund Roughton, Demonstrator of Anatomy at St. Mary's. The tape indicated a diminution of 1 in. distance from the first dorsal to the fourth lumbar spine when suspended from the occiput and chin alone, and there was a further decrease of $\frac{1}{4}$ in. in this region when support from the axillæ was added. The latter fact is very significant, and it agrees entirely both with what may be seen on the dead body, and with the views on the subject of muscular action, which shall be stated presently. Mr. Roughton was well satisfied of the accuracy of the figures.

It is difficult to obtain a subject sufficiently tolerant of the position to allow good measurements to be taken while suspended from the occiput and chin, and there remain the data in but one other case, that in which the writer suspended a colleague. There occurred then a contraction in the dorso-lumbar curve of $\frac{1}{4}$ in., an increase in the cervical apparently of less than $\frac{1}{4}$ in., and a total contraction of $\frac{1}{8}$ in. In the writer's own case, when suspended by the head alone, the total length of the column was unaltered, an elongation of 1 in. in the cervical compensating for a similar contraction in the dorsal district. When again suspended both from the head and axillæ the dorsal shortening was $\frac{1}{4}$ in. more, while the length of the neck remained the same, *a total shortening* then in this instance *of one quarter of an inch.* In these three cases, therefore, there was a total shortening throughout the column of $\frac{1}{8}$ in. to $\frac{1}{4}$ in. *A shortening of the dorso-lumbar curve was invariable,* ranging from $\frac{1}{4}$ to $1\frac{1}{4}$ in., and averaging nearly 1 in. It was judged, moreover, that the lumbar curve was in all cases unaffected. The result of the re-

maining three cases was generally the same. In all the dorso-lumbar line was shortened, with the single exception alluded to. In all except this instance a shortening of the whole column was found. In only one case was there apparent shortening of the neck. But it has been said that no positive statement can ever be made upon that point. One fact only has been ascertained beyond doubt, namely, that the neck is less elongated by the process in those who have been several times suspended than in those who are submitted to the operation for the first time.

The uncertainty of this class of measurement impressed me so strongly that I determined to make others under like conditions in the dissecting room, and on the dead body. These were necessarily quite accurate. The muscles were removed from the back—the bony points cleaned and exposed to view—and ample time was available to carry out the experiment with precision. The measurements obtained in this way tallied remarkably with the average of those taken during life. Where they differed the discrepancy was referable to the uncertainty already alluded to as to what position of flexion or extension should be given to the head in the original estimate. The results are instructive in many particulars, and they are appended.

Dissecting room, St. Mary's Hospital, October 26th, 1889.

CASE 7.—Body suspended. The limbs had been removed, and the thorax and abdomen dissected. The dorsal muscles were carefully cleaned off, all the ligaments remaining. The body was then suspended from the chin and occiput and the first series of measurements along the spines was taken. Three heavy bricks (17 lbs.) were then attached to the pelvis while the body hung, and the spines were measured a second time.

a. Before adding weights.

i. Upper border of 2 C. to lower border of 1 D. = 4½ in.
ii. Lower border of 1 D. to lower border of 1 L. = 10½ in.

b. After weights were added.

i. As before = $4\frac{1}{16}$ in.
ii. As before = $9\frac{15}{16}$ in.

Therefore there was a stretching in the cervical region of $\frac{5}{16}$ in., and a contraction in the dorsal of $\frac{1}{4}$ in.; a total stretching of $\frac{1}{16}$ in.

The anterior measurements were not taken, but it was apparent that the anterior common ligament was greatly extended. The cervical and dorsal curves were nearly abolished. The splanchnic nerves were stretched like fiddle-strings.

An incision was made separating the anterior common ligament and detaching the intervertebral disc beneath the third cervical vertebra. The aperture did not gape, though the weights remained on.

The body was then taken down and the laminæ and pedicles removed, while the body was supported on the table by a block under the thorax. As disclosed in this position, the cord and nerve-roots were stretched tense. The body was then hung up as before with weights. The cord enclosed in dura mater then bulged out towards the back, being very loose and relaxed. The dura mater was wrinkled transversely. The course of the nerve-roots from their origin to the intervertebral foramina was much shortened, and they were in sinuous curves. This effect was equally pronounced in every region. In the cervical region, as elsewhere, the apparent origin of each nerve was well above its point of exit from the spinal canal.

Dissecting Room, St. Mary's Hospital, November 6th.

Cases 8 and 9.—Body suspended. The lower limbs were attached, but the feet had been removed. The arms were off. The brain was removed. The thorax and

abdomen were dissected. The dorsal muscles were cleaned off and the spines and laminæ exposed. Anterior and posterior measurements were taken along the bodies and spines—(1) with the body on the table; (2) suspended by occiput and chin; (3) two heavy bricks (11½ lbs.) being attached in the latter case.

I.—A. Anterior measurement (body lying on its back on the table and extended to the utmost):

(1) i. From basilar pr. to lower border of 1 D. . = 7½ in.
ii. From lower border of 1 D. to lower border of 5 L. = 16½ in.

B. Posteriorly (on table):

(1) i. From tubercle on atlas to lower border of 1 D. = 6 in.
ii. Dorso-lumbar as before . . . . = 18½ in.

II.—A. Anteriorly:

	(2) Simply suspended.	(3) Weights added.
i. As before ...	5⅞ in. ...	6 in.
ii. As before ...	16½ in. ...	17 in.

B. Posteriorly:

i. As before ...	5⅞ in. ...	5⅞ in.
ii. As before ...	18½ in. ...	18½ in.

Analysis.—Anteriorly: The discrepancy of 1⅛ in. between the first and second measurements shows that a suitable position of the neck had not been obtained, and indeed, this can never be made certain of. Dorsal stretching ⅜ in. The addition of heavy weights stretched the cervical region only ¼ in., and the dorsal ½ in. more. Posteriorly: Cervical region contracted ¼ in. when suspended first, and this underwent no change when weights were added. Dorso-lumbar first stretched ¼ in., and again contracted to previous length.

(*a*) Body suspended simply:

Cervical ? } anteriorly.
Dorsal extension ⅛ in. }
Cervical contraction ¼ in. } posteriorly.
Dorsal extension, ¼ in. }

(*b*) Weights added:

Cervical extension ¼ in. } anteriorly.
Dorsal extension ¼ in. }

Cervical unaffected } posteriorly.
Dorsal contraction ¼ in. }

The value of these experiments depends on the fact that they were done under conditions which entirely got rid of muscular tension. They represent the effect of suspension on the osseo-ligamentous skeleton stripped of its coverings. The bony points were exposed, and measurements were made under the most favorable circumstances. The same uncertainty as before attaches to figures which deal with the posterior surface of the neck, but in a less degree. Those taken in the dorsal and lumbar region are absolute and reliable.

In the first experiment (Case 7) the addition of weights to the body already suspended caused a shortening along the summmit of the dorsal and lumbar spines—a shortening in this case of ¼ in. Under the same circumstances the cervical spines were separated by a total distance of $\frac{5}{16}$ in., the length of the column remaining unaltered. The measurement of the cervical region in this case, being made under like circumstances before and after the addition of weights, is presumably correct. At the same time the movements of the vertebræ amongst themselves could be watched. The cervical and the dorsal curves straightened out by extension of the posterior ligaments above, and of the anterior ligaments below, and by separation of the bodies below. The appearance of the dura mater when the spinal canal was opened under these circumstances will be referred to later.

In the second experiment (Cases 8 and 9) the anterior measurements were taken as well as the posterior. The results of the latter were in the main those obtained under all other conditions, but modified in a very interesting manner. The line along the dorsal and lumbar spines was stretched ¼ in. when the truncated and eviscerated body was simply suspended. When, however, a

FIG. 2. Compounded from two photographs of dissected and dried specimen (exhibited). The specimen was suspended first simply and afterwards with 18 lbs. added below. Two strings were stretched horizontally in front of the specimen. In the first photograph the strings crossed the points A A, in the second the points B B. B B is a greater distance than A A. This shows a slight relaxation of the spinal cord which is exposed. The specimen is from the museum of St. Bartholomew's Hospital, and it was dissected by Mr. Roughton.

weight of 11¼ lbs. was added, the line contracted to its original length. There was thus no elongation, but rather, perhaps, a shortening. The ¼ in. gained on suspending the body is very likely the expression of an error due to the fact that the unsupported vertebral column collapsed a little when laid on the table, its curves straightening and the spinous processes in the dorsal region approaching one another in a somewhat concentric manner. This probably explains another anomaly—the apparent shortening of the spinous surface in the neck, which stretched again to its original length when weight was added. The collapse of the curve in the cervical region when the body lay on the table would have the effect of expanding the spines in a fan-like manner. The weight of the body was sufficient to overcome this malposition, and the further weight of 10¾ lbs. acted not by stretching the posterior ligaments, as was the case with the body prone, but by compression of the intervertebral discs anteriorly. This is a fact of great importance, and will be referred to again. The total result was to leave the spinous surface unaffected when the light body was suspended and a subsequent contraction of ¼ in. when weight was added. All this contraction, as before, occurred in the dorsal region.

The results of the anterior measurement are very suggestive. The basilar process having been taken as the upper fixed point for the cervical region makes one series of figures unreliable. Uncertainty as to the proper degree of flexion of the head is the cause, and the doubt is illustrated by so great a discrepancy between the length along the vertebræ when the body was on the table and when it was simply suspended—a discrepancy of 1⅜ in. The comparison in this case, therefore, does not hold. When weight was added to the suspended body the cervical curve expanded ¼ in. In the dorsal and lumbar regions together the effect of suspension was to stretch the anterior surface of the column ⅜ in., and when weights were added a further stretching of ½ in. took place, a total elongation of this aspect in the weighted body of ⅞ in. This appeared

to come exclusively from the dorsal curve. If it be remembered now that the line of the spines at the same time shortened, it will appear that the effect of suspending the weighted body was to straighten out the dorsal curve by a movement of the vertebræ each around an axis which corresponds to the situation of the posterior border of the bodies. The anterior common ligament was stretched, the

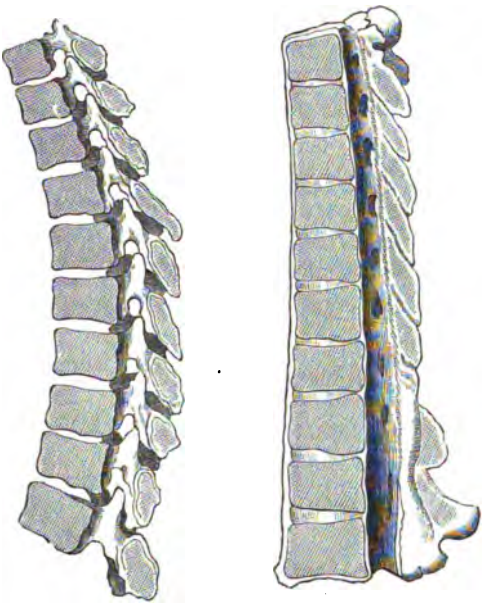

FIG. 3. The lower part of the thoracic curve, with spinal canal exposed. The left-hand figure is drawn from the skeleton, the right-hand figure from a specimen which was forcibly straightened.

ligaments of the post-neural segments were relaxed, and the spinal canal, since it lies behind the axes of rotation, must have been shortened. At the same time the intervertebral foramina are approximated.[1] From both facts it follows that the spinal cord would be relaxed, and most on

[1] See the writer's paper on "The Disposition of the Vertebral Column in Hanging and Swinging Postures," 'Journ. of Anat. and Physiol.,' 1890.

its dorsal surface (Fig. 3); the nerve-roots would be relaxed with it, and still more so by the approximation of the intervertebral foramina by which they leave the spinal canal.

The cervical curve was stretched anteriorly ¼ in. and probably about as much between the spines. The curve tended to straighten by overlapping of the laminæ in front. It would seem probable that so slight a change does not in any way affect the cervical portion of the cord, which besides is disposed to accommodate itself to the freest range of movement. The changes that occur are to some extent compensatory; for while the absolute elongation of ¼ in. on both back and front would tend to render the dura mater, if not the cord, tense, straightening of the curve will act in the opposite direction, since the cord lies in its concavity. My own belief is that it is impossible in this way to stretch the dura mater without dislocating the neck. This view is strongly confirmed by observation of the cord *in situ* with the spines and laminæ removed.

The absence of the soft parts would obviously modify the conditions of extension or relaxation. Here, therefore, are tabulated the measurements in two cases taken in the post-mortem room without dissection.

November 12th, 1889. *Post-mortem Room, St. Mary's Hospital.*

Case 10.—Body of a man, æt. 35, who died of Bright's disease.

1. Anterior measurements:

	Lying prone after long suspension, about 2 hours.	Suspended.
i. 2 C. to 1 D.	5¼ in.	5¼ in.
ii. 1 D. to 12 D.	9¼ in.	10 in.
iii. 12 D. to 5 L.	7¼ in.	6¾ in.

2. Posterior measurements:

	Prone before suspension.	Suspended.
i. 2 C. to 1 D.	3¾ in. (5)	3½ in.
ii. 1 D. to 12 D.	11½ in. (12)	10½ in.
iii. 12 D. to 5 L.	4¾ in. (4¾)	4¾ in.

The figures in brackets indicate measurements taken when a block was under the thorax. The others give results without the block. This body was suspended without the viscera; and the cranium had not been opened. While suspended the spinal arteries were injected from the following trunks: abdominal aorta in two places, above and below the cœliac axis; right vertebral, left vertebral, and left ascending cervical. It was cut down after two hours and the entire cord removed This is specimen marked Cord A.

Analysis.

Anteriorly:

Cervical	stretched ½ in.
Dorsal	„ ½ in.
Lumbar	contracted ½ in.[1]

Posteriorly:

Cervical	stretched ½ in.
Dorsal	contracted ½ in.
Lumbar	unaffected.

The significance of these tables is modified by the fact that the first measurements (anteriorly) were taken after two hours' suspension. They indicate that a certain resilience will occur after death. As an indication of the effect of suspension the difference in the anterior measurements is probably too small.

Without making allowance for this, the gross result is:

Anteriorly:

Cervico-dorsal region	stretched ½ in.
Lumbar „	contracted ½ in.

Posteriorly:

Cervico-dorsal region	contracted ½ in.
Dorsal region alone	„ ½ in.

[1] This was repeatedly verified. The fact is very significant.

November 12th. *Post-mortem Room.*

CASE 11.—Body of a boy, æt. 15, who died of typhoid fever.

1. Anterior measurements:

	(a) Lying prone on table.	(b) Suspended.
i. 2 C. to 1 D. ...	4½ in. ...	4½ in.
ii. 1 D. to 12 D. ...	8¾ in. ...	9½ in.
iii. 12 D. to 5 L. ...	6½ in. ...	6½ in.

2. Posterior measurements:

i. 2 C. to 1 D. ...	4½ in. ...	3½ in.
ii. 1 D. to 12 D. ...	9¼ in. ...	9½ in.
iii. 12 D. to 5 L. ...	5½ in. ...	5½ in.

In measuring the cervical region the head was supported in a position judged to be the same as that assumed in suspension, and it was drawn outwards. This body had the brain and viscera removed before suspension. The weight was supplied by attaching 11 lbs. to the legs.

Analysis.

Anteriorly:

Cervical region unaltered
Dorsal „ stretched ½ in. } stretching ½ in.
Lumbar „ unaltered

Posteriorly:

Cervical region ?
Dorsal „ contracted ¼ in. } contraction ¼ in.
Lumbar „ unaffected.

The experiments, of which a detailed account has been given, appeared to furnish collectively all the available data. They comprise measurements taken under three classes of circumstances: namely, those upon the living body, those upon the dead body with the bony points and ligaments exposed, and those upon the dead body undissected. Each of the series is instructive in its way, and each tends to illustrate the others. One fact stands prominently forward. So far from finding the great elongation of the dorsal region of the spine reported by others, I have only once, either in the living body or in the dead,

met with any lengthening at all, and that was in a case of lateral curvature. The remarkable consistency amongst the results in this particular goes to prove that the same forces are at work upon the vertebral column, alike in the living and the dead body. In the former case there are other forces added, *e. g.* the effect of muscular tension, which remains to be considered. The posterior measurement, that along the summits of the spines, is the only one which can be obtained under all conditions.

An analysis of the eleven experiments recorded would show *that the result of suspension is to cause an average extension posteriorly in the cervical region of* $\frac{1}{10}$ *in., a contraction in the dorsal region of more than* $\frac{1}{8}$ *in.*, no effect upon the length of the lumbar curve, and *a total contraction of rather less than* $\frac{1}{8}$ *in., throughout.* Anterior measurements can be had only on the dead body. Averages compiled from the tables show an *increase of* $\frac{1}{8}$ *in.* in the cervical region, of $\frac{1}{16}$ *in.* in the dorsal, and of $\frac{17}{48}$ *or* 1 *in.* in the two taken together. At the same time there is a diminution of $\frac{1}{4}$ in. in the lumbar curve.

Something has already been said of the manner in which these effects are produced, but they need a little further consideration. The statistics show that the chief result of suspension on the dead body, is a straightening out of the cervical and the dorsal curves. But in the two districts this takes place in a different way. In the dorsal region, where the convexity lies behind, straightening takes place by lengthening of the anterior surface (Fig. 3). The anterior common ligament and the marginal attachment of the intervertebral discs give way, and the bodies separate to the extent, on an average, of $\frac{1}{16}$ in. At the same time the spines approach one another. There is consequently a point of rotation somewhere between. This is doubtless in the situation of the posterior border of the bodies of the vertebræ, which are thicker there than in front. In the cervical curve the case is different. Not one or two, but several strong ligaments are present to resist the stretch-

ing of the concavity which occurs readily enough lower down.[1] On the other hand, the bodies of the vertebræ are not thicker towards the convexity in front, but the intervertebral discs are thick in that situation, and indefinite compression is possible. It is probable that it takes place, and that the curve tends to straighten out at the expense of its anterior surface—the convexity—which actually shortens. The difficulty of making accurate measurements in the neck renders it impossible to show this point in figures, but in addition to the anatomical facts mentioned, there are others which support the view: the remarkable lip-like projection on the lower border of each body appears calculated to guide the upper border of the body below in the direction required, and finally some such appearance is presented when the dissected body is suspended. Again, the fact recorded in one of the dissecting-room experiments, when the anterior common ligament was separated and the incision did not gape, lends countenance to the view. Beyond this, however, there is the fact that actual shortening of $\frac{1}{4}$ in. to $\frac{1}{2}$ in. does occur in the lumbar region in which the curve is similarly situated, and there also it must be by compression of the intervertebral discs in front. In the cervical region the spinal cord occupies the concavity of the curve. If, then, this straightens out without separation of its extremities, the cord is relaxed thereby. The process which we have traced in the dorsal region is obviously attended with shortening of the spinal canal and consequently relaxation of the cord in that situation. At the same time the approximation of the intervertebral foramina still further shortens the nerve-roots. This, I believe, is the process in the dead body.

It remains to consider the effect of muscular tension, —and first where the body is suspended from the axillæ in the usual manner. Suspension from the axillæ, in so far as it is effective, is suspension from the scapulæ and clavicles, and ultimately from the muscles which

[1] Op. cit., 'Journ. of Anat. and Physiol.,' 1890.

connect those bones with the trunk. These muscles may be divided into two classes: first, those (the trapezius and rhomboids) which take origin from spinous processes; secondly, those (serratus magnus, and pectorals) which arise from the ribs or some part in front of the vertebræ.

Muscles of the first class tend to approximate the spines directly, and so aid the action of gravity as seen in the dead body (Fig. 4, B). Again, the muscles which arise from ribs do so too, because they raise the ribs which are firmly attached to the transverse processes by the costo-transverse ligaments, while they are free to move on the articulation

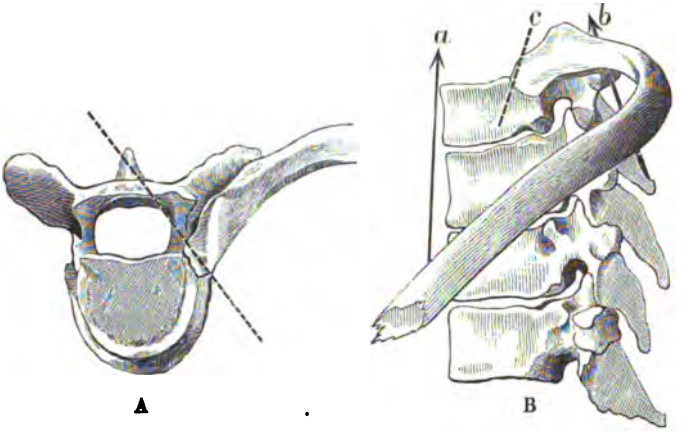

FIG. 4. To show the axis of rotation and the point of application of muscular force in the dorsal region.

A. The dotted line indicates the axis of rotation under the influence of muscular action.

B. *a*. The direction in which the muscles act which are attached to the ribs; *b*. The direction of muscles attached to spinous processes; *c*. The axis of rotation.

of their heads with the bodies of the vertebræ. They constitute, therefore, a set of levers of the second order, the weight being at the transverse processes. Since, however, the transverse processes are situated behind the axes of rotation mentioned above (Fig. 4, A), muscular tension in this case also acts in the same direction as gravity, and with it helps to relax the spinal cord by shortening the spinal

canal. This view is further supported by figures. When the writer was suspended from the head by Mr. Roughton there was a contraction of 1 in. posteriorly in the dorsal region, and when support was given from the axilla a further contraction of ¼ in. ensued.

The question of muscular tension in the neck is more complicated, and that of forced muscular action still more so. When a patient is first suspended, especially if it be by the head alone, he commonly hangs with the body thrown back, so that the legs are well behind the plumb-line from the point of support. At the same time the back is curved in the direction of opisthotonos. This is an effect of muscular action, and it originates in an involuntary effort to resist straightening of the cervical curve. For this purpose the muscles of the neck contract powerfully. So do the erectores spinæ and their continuations in the back. Under these circumstances the maximum of posterior shortening occurs, and doubtless also the greatest possible elongation along the bodies of the vertebræ in front. It is then that syncope, vomiting, and diarrhœa have followed suspension. These may find their explanation in the stretching of the splanchnic nerves, which I have repeatedly seen on the dead body. Whether this be so or not, I am convinced that the splanchnics are the only nerve-structures which *are* stretched.

It appears, at all events, that the spinal cord and its nerve-roots are not stretched but relaxed. This conclusion results alike from measurements, from *a priori* anatomical considerations, and from actual demonstration on the dead body with the spines and laminæ removed.

Is it possible to attribute to this relaxation any of the curative effect of suspension? Doubtless it is. Those who have assumed that stretching of the cord might be competent to break down adhesions and overgrown neuroglia have forgotten important facts of anatomy. The dura mater is a highly inextensible membrane; it is connected to the bony walls which enclose it, not only above and below, but also by means of strong processes

to each pair of intervertebral foramina. Any movement which it admits is checked in this way, and it cannot be extended indefinitely from one segment to another. The most effectual way to stretch the dura mater is to bend, not to straighten, the spine, as can be shown when the cord is exposed *in situ*. Again the movements of the cord within the dura mater are controlled both by the connection of the nerve-roots with the latter at the intervertebral foramina, and by the processes of the ligamentum denticulatum. Finally the vertebral column is admirably constructed to defend the cord from the action of excessive force such as it has been attempted to exert. But how would extension of the cord, if it were possible, affect a patch of sclerosis? Injuriously if at all. Tension on a rope does not tend to loosen its fibres; but active relaxation, as by doubling up the rope, does. The white matter of the cord is formed of strands of nerve-fibres derived from the nerve-roots. In sclerosis, these are shrunken, compressed, and cemented together. Adhesions amongst the fibres will be broken down, if indeed they can be broken down mechanically—best by relaxation, such as it is contended occurs. This may be a part of the *modus medendi*, but far more efficacious must be the concomitant effect on the spinal blood-vessels and lymphatics. Sclerosis is associated with and maintained by mal-nutrition. The vessels are thickened and choked by pressure from the contraction of surrounding fibrous tissue. If this be opened up and relaxed, more blood will enter. The posterior columns will be flushed. There is a possibility that, with freer circulation, morbid products will be removed, regeneration of tissue promoted, and suspended function restored. I have injected spinal cords while the body was suspended. The naked-eye appearances favour the view stated here and it is hoped that microscopical sections will throw further light on the subject. Some of these are now in course of preparation and others are exhibited. It must not be forgotten, however, that there remains the possibility that vital changes, not demonstrable

on the dead body, have their share in the result. Allusion has been made to the stretching of the splanchnics. When this is excessive, as it most probably is in those cases where the body is curved backwards, stretching may cause a temporary paralytic lesion, whence possibly arises pain in the epigastrium, syncope, vomiting, diarrhœa, and, it may be, death. A slighter degree of extension might constitute an irritative lesion, and it is conceivable that such would have its *rôle* in the therapeutics of suspension.

The conclusions to which I have been led may be briefly summed up.

1st. A stretching of the cord would give no rational explanation of the effects sought, but this stretching does not occur.

2nd. A considerable and effective relaxation, both of the cord and its nerve-roots does occur in suspension. Relaxation is competent to account for the benefit sought. It takes place to the greatest extent in the dorsal curve, where also the tabetic lesion is always and chiefly situated, and more in the posterior than in the anterior columns of the cord.

3rd. This effect is produced by the weight of the body alone, and is aided by muscular tension when the body is suspended from the axillæ.

4th. Muscular tension, like the force of gravity, acts beneficially most, if not only, in the dorsal region. *Relaxation of the cord in the neck is impossible, and cervico-occipital suspension is not only dangerous and unpleasant, but unscientific and inoperative.*

5th. It follows, therefore, that measures should be directed towards the dorsal region alone. For the old and infirm, the present method of suspension from the axillæ, but from the axillæ alone, will probably remain the best. No strain should ever be put upon the head. If relaxation of the cervical portion of the cord be aimed at, this can be obtained in no way better than by poising the head upon the vertebral column in the natural position of ease. If these views are correct, the best plan of all,

where it can be adopted—as in younger men—would be found in a judicious course of gymnastics, which should have for their object a moderate and associated contraction of the muscles of the back combined with tension of those which connect the scapula and clavicle with the trunk. Simply to hang by the arms from a point above the head for a short time would probably be attended with better consequences than are attained by the use of the cumbrous apparatus at present in vogue.

(Since writing the last paragraph I have made trial of this plan, and in two cases I have obtained results not less remarkable than those published elsewhere.)

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 53.)

A CASE OF HERNIA OF THE CÆCUM,

ENTIRELY WANTING IN A PERITONEAL SAC,

IN WHICH

STRANGULATION AT THE INTERNAL ABDOMINAL RING CO-EXISTED WITH AN INTUSSUSCEPTION THROUGH THE ILEO-CÆCAL VALVE.

BY

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THE following communication is, I regret to say, necessarily imperfect, inasmuch as it was impossible, although the patient died, to obtain a post-mortem examination of the parts involved. At the same time I trust that the rarity of the case described will be sufficient to justify its publication.

On the evening of October 26th, 1889, W. K—, a labourer, æt. 52, walked leisurely into St. George's Hospital complaining that an old-standing rupture, with which he was afflicted, and which he had been previously able at all times to replace with ease, had become irreducible.

The hernia, he said, had existed for eighteen years, but he had worn a truss during the last two years only, and

even during that period with much irregularity. The truss when in use kept the rupture up, as a rule; and if by chance it came down behind the instrument, reduction was easily effected, being always accompanied by the ordinary and characteristic "gurgle."

Upon inquiry it was further elicited that the scrotum was always considerably larger and harder than normal on the affected side, in spite of the apparent completeness of the reduction of the hernia.

Twenty-four hours before the patient came to the hospital (the rupture "being up" at the time, to the best of his knowledge, although the truss was not in use) he noticed, whilst lifting a heavy basket, some discomfort about the scrotum, of a kind he had never before felt.

Upon examination he found that the rupture was larger than he had previously seen it, although he had experienced none of the usual sensations warning him of a sudden descent of gut.

In spite of this he continued his work, the size of the hernia gradually increasing in a manner quite different from anything he had before noticed. This gradual descent and steady increase of the rupture are significant facts, as will be subsequently seen.

Considerable pain of a burning nature soon supervened, which was entirely confined to the scrotum. He was therefore compelled to leave his work. He returned home, and having failed to reduce the tumour himself in the ordinary way he called in a medical man, who after repeated attempts was equally unsuccessful. Shortly after this he vomited. The night which followed was restless, and vomiting occurred at frequent intervals up to the time of his coming to the hospital. The bowels acted for the last time on the morning of the 24th.

The house surgeon, finding that the hernia was obviously irreducible and strangulated, at once sent the patient to bed. The buttocks were well raised, and ice applied to the rupture.

In spite of this proceeding the tumour very slowly

increased in size, and I was summoned to see the man soon after midnight (about six hours after his admission), when I found the condition of things as follows.

The man was stout and "full-blooded." The expression was somewhat anxious, the skin moist, and the pulse quick, regular, but inclined to be small. The temperature was subnormal. There was an entire absence of pain, umbilical or otherwise. There was nausea, but no vomiting.

The right side of the scrotum was occupied by a pyriform swelling, about the size of two closed fists, which ran upwards along the inguinal canal. This tumour was dull and full, but not extremely tense. There was no impulse on coughing. There was no abdominal distention, the parietes were flaccid, and no tenderness of any kind was present excepting at the extreme upper limit of the scrotal swelling.

Although the case appeared to be without doubt one of strangulated hernia, there was something about the general aspect and feel of the tumour which was not exactly like anything of the same kind which I had previously seen, so much so that I told Mr. Higgins, my house surgeon, that I fully expected to find an unusual state of affairs upon performing herniotomy, which was clearly necessary.

The patient having been anæsthetised, I cut down upon the hernia in the ordinary way, and ultimately exposed a somewhat tense tumour, obviously containing fluid, which at first sight seemed likely to be the sac of the rupture, although it had not the appearance of such as commonly seen. Upon pinching up this apparent sac there could be felt inside it a mass which slipped away from between my fingers, precisely as a tense knuckle of gut often does when felt in this manner inside a not very greatly distended peritoneal sac.

Although I was in much doubt about the structure exposed being the sac, for it struck me at the time, and I so stated my feeling to those present, that the tumour was not unlikely to be gut which was uncovered by peri-

toneum, I decided to open it; for not only was it entirely irreducible in spite of free division of all surrounding soft parts which could be divided apparently with safety, but the mass, which could be felt inside, led me to suspect the existence possibly of a polypoid tumour of some kind, if the structure with which I was dealing proved to be actually bowel.

I must, however, confess that the possibility of the mass being an intussusception did not occur to me at this time.

A small incision having been made into the tumour, there spurted out with some force a quantity of clear watery fluid precisely like that which commonly escapes upon opening an ordinary sac in the course of herniotomy.

This, for the moment, made me think that my apprehensions were unfounded, and that after all it was merely a peritoneal sac that had been opened.

The walls of the tumour, however, as its contents escaped, were seen to contract *actively;* moreover, the last portions of the fluid discharged were opaque, and had a fæcal smell; finally, there could be seen protruding from between the lips of my little incision some mucous membrane.

It was clear, therefore, as I had half anticipated, that I had opened the gut. It was equally obvious, as will be presently seen, that the gut was quite devoid of a peritoneal investment.

As the mass, which has been mentioned as having been felt inside, remained unchanged, the incision in the bowel was freely enlarged, and the following conditions of parts revealed.

The structure which had been laid open was the cæcum. Projecting from the upper and inner part *through* the ileo-cæcal valve, the margins of which could be felt grasping its base, was an intussuscepted piece of the ileum about three inches long.

Passing from the upper and outer part of the herniated cæcum could be seen the opening of the colon, which was

tightly constricted by a band of tissue crossing it at the internal ring. At the inner part could also be made out the opening of the vermiform appendix, the lumen of which seemed to pass upwards and backwards.

The calibre of the intussusception was so small, in consequence apparently of external constriction, that it would admit a single finger only with some difficulty.

After the division of the tight band which crossed the hernia at the internal ring, and which had escaped my notice before opening the gut, the bowel generally was so much liberated that the lumen of the colon appeared of normal size, and the intussusceptum admitted three fingers with ease, a quantity of flatus and a little fæcal matter being expelled at the same time.

Upon turning up the cæcum, which was of course now flabby and collapsed, the most careful examination failed to detect anything upon the surface of the gut which was in the least degree suggestive of the presence of peritoneum; but behind it there was seen passing down into the scrotum, and adherent to the testicle, a slender, rather œdematous piece of omentum, lying in a perfect and rather thin peritoneal sac, which, there is little doubt, had also contained the original hernia which the patient had been in the habit of reducing from time to time.

Upon passing the finger upwards, behind the cæcum, along the surface of this sac, it was arrested just at the point from which the appendix seemed to spring, where all the parts appeared hopelessly matted together.

The intussusception which showed, after the division of the constricting band mentioned, very little indication of congestion, and was but slightly swollen, was quite irreducible. There seemed also not the least chance of the hernia itself being made in any way reducible, excepting by the performance of extensive abdominal section, which the desperate condition of the patient at this time rendered entirely unjustifiable. The only proceeding available, therefore, was to suture the edges of the wound in the bowel to the margins of the scrotal incision—making,

in fact, a temporary artificial anus with a view to abdominal section, reduction of the gut, and restoration of its canal on a future occasion, if the patient should rally sufficiently—an occurrence which appeared highly improbable.

The existence of the intussusception entailed of course, under these circumstances, no danger of obstruction, for if it were not sufficiently patulous to allow spontaneous evacuation of the intestinal contents, the removal of any flatus or fæcal material which might accumulate could be easily effected by the passage of a tube through the invaginated gut.

For twelve hours after the operation the patient rallied to some extent, but did not gain sufficient strength to justify further operative measures.

Subsequently he gradually sank and died, apparently of asthenia, at 3 p.m. on October 29th.

The operation was followed by no abdominal distention, the parietes remained quite flaccid, and there was neither pain, tenderness, nor discomfort of any kind.

Nourishment was taken freely, and from time to time a little flatus and feculent matter came from the intussusception, which was perfectly patulous.

On the evening of October 28th, in order to make certain that no accumulation was taking place, a long tube was passed through the invaginated bowel, and about a quarter of a pint of liquid fæces withdrawn.

Remarks.

I. *As to the herniated cæcum.*—The occurrence of hernia of the cæcum without a peritoneal sac appears to have been formerly accepted by common consent as a fact which in itself was not in any degree remarkable.

It was also, I believe, rather extensively taught, even up to comparatively recent times, that on this account cæcal hernia was frequently irreducible.

Recently, however, the researches of Treves and others tend to show that the peritoneal relations of this viscus are such that *extra-peritoneal* hernia of the cæcum must of necessity be so rare that the possibility of its existence is hardly worth consideration.

This view is strongly supported by the absence hitherto, so far as I can ascertain, of a precise record of any instance of cæcal hernia devoid of a sac, which has actually been met with in practice.

A careful search through the notes of 565 cases of strangulated hernia, successively recorded in the register of St. George's Hospital, fails to afford a single instance of this condition, although several varieties of cæcal hernia occur.

Another point of some interest, in connection with the subject under discussion, shown by these 565 miscellaneous cases is the sigularly small number of examples of strangulated herniæ of all kinds in which the cæcum formed any part of the hernial tumour, for in these 565 cases the cæcum was present in nine only (that is, 1·59 per cent.); and of these nine instances all were of a complicated nature excepting two, one of which was a remarkable case in which the cæcum occupied a hernial sac in the *left* groin.

Some further particulars of these nine cases will be found in the accompanying table (p. 138).

It will be seen that a complete peritoneal sac existed in all, with the exception of the one now recorded. In the whole series of 565 miscellaneous herniæ there was no case in which the contents of the sac were wanting in a complete peritoneal investment.

The case I have described in this communication is without doubt a genuine instance of the extra-peritoneal form of hernia; for, as has been pointed out, there was not a vestige of peritoneum or anything approaching to it in appearance, either in the form of a sac or as an immediate visceral investment.

The escape of clear watery fluid, when the gut was

opened, is worthy of attention, since it is an unusual occurrence which might at any time be liable to mislead. The presence of such a fluid was, I presume, caused by excessive secretion from the congested and swollen mucous membrane in the tightly constricted bowel, which, at the time of strangulation, was free from fæcal contents.

I have had no previous experience of clear watery fluid coming from a knuckle of strangulated gut, but I have seen in the post-mortem room a case in which the vermiform appendix was greatly distended by a precisely similar fluid, in consequence apparently of its opening into the large intestine having been blocked. Instances of the same kind have been observed, I have no doubt, by others.

II. *As to the intussusception.*—This afforded a fair example of intussusception of the ileo-colic variety, *i. e.* invagination *through* the ileo-cæcal valve, which, according to the accepted authorities, is met with in only 8 per cent. of all cases of this affection.

An interesting point here arises with reference to the relation of the intussusception to the production of the hernia, and *vice versâ*.

The general aspect of the invaginated bowel, the disappearance of all congestion after the division of the external constriction, together with its patulous state, and the absolute irreducibility, are facts which seem to point to the condition being chronic.

On the other hand, the very gradual descent of the rupture, the unusual kind of pain, and the general sensations experienced, which were just such as might have been caused by a piece of gut making its way through loose connective tissue, point to the probability of the hernia being of a different kind from that which had previously troubled the patient.

This probability was converted into something very like a certainty by the discovery of the old and independent sac containing adherent omentum, but no bowel, which is referred to in the description of the case.

The evidence in this respect, therefore, seems to indicate that the presence of the cæcum in the hernial tumour was quite a recent condition.

At the same time it is, of course, fair to admit that the cæcum might possibly have occupied the same position previously without giving rise to any noticeable symptoms until the state of affairs became altered by the occurrence of the intussusception.

III. *As to the case generally.*—Under this head there is little to be said. Taking all the circumstances into consideration, the case is the most complicated example of strangulated hernia which in the course of a considerable experience I have yet had to deal with.

The treatment adopted seems to have been the only rational proceeding which was available. The issue from the first was hardly doubtful, and the death from asthenia was only what could have reasonably been expected.

Finally, it appears to me that, setting aside certain minor points of interest, the co-existence in this patient of two such conditions as extra-peritoneal hernia of the cæcum and the least frequent form of intussusception (ileo-colic) presents a case which, if it is not unique, as I suspect it to be, must at least be of such singular rarity that I have ventured to bring it to the notice of this Society.

Table of the only cases in which the Cæcum was found to form any portion of the Hernial Tumour in a series of 565 *Herniotomies described in the Records of Strangulated Herniæ at St. George's Hospital.*

No.	Age.	Sex.	Side.	Variety.	Contents of sac and their condition.	Result.	Remarks.
1	14	M.	Right	Congenital inguinal	Commencement of large gut and end of ileum—vermiform appendix (sloughing and perforated). Tight stricture caused by adhesions to independent piece of small intestine which crossed the sac	Died	Patient was in hopeless state from collapse and suppurative peritonitis at time of operation.
2	54	M.	„	„	The tunica vaginalis which formed the sac had been ruptured by an accident, and through the rent protruded the cæcum and a quantity of small intestine. Stricture at edges of rent	Recovered	—
3	73	M.	„	„	Bloody fluid. Testicle, small intestine, cæcum, and appendix all matted together	Died	Patient in condition of almost hopeless prostration.
4	54	M.	„	Inguinal	Pus, gangrenous vermiform appendix. Tight stricture at internal ring	„	Patient in condition of almost hopeless prostration.
5	70	F.	„	Femoral	Large mass of adherent omentum, cæcum with its appendix, ileum, and part of transverse colon	„	Patient in condition of almost hopeless prostration.
6	48	M.	Left	Inguinal	Sanious fluid. Vermiform appendix, cæcum, ileo-cæcal valve, and some small intestine. Very tight stricture	„	—

7	58	M.	Right	Congenital inguinal	Cæcum and large mass of strangulated omentum	Recovered	Patient in good condition at time of operation.
8	2½	M.	„	„	Clear fluid. Whole cæcum and appendix, ileo-cæcal valve, and one inch of ileum	„	Patient in good condition at time of operation.
9	52	M.	„	Inguinal	Hernial tumour contained cæcum (without peritoneal sac or investment). Ileo-cæcal valve with ileo-colic intussusception. Sac of another hernia containing adherent omentum	Died	Case now recorded.

The principal points indicated by the above table are (1) the comparative infrequency of strangulated hernia of the cæcum in any form, the number of cases met with in this large series of 565 herniotomies amounting to only 1·59 per cent.; and (2) the extreme rareness of *uncomplicated* cases of this form of strangulated hernia, two instances only being found in this same series, *i. e.* ·35 per cent.

Case No. 9 (now recorded) stands alone.

The very high rate of mortality (66·6 per cent.) is clearly due to the critical condition of the majority of the patients at the time of operation.

The cases here tabulated are exclusive of one instance in which the relation of a gangrenous cæcum to a hernial sac was doubtful.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 56.)

RHEUMATISM, ITS TREATMENT PAST AND PRESENT;

WITH SPECIAL REFERENCE TO RECENT EXPERIMENTAL RESEARCH ON SALICYLIC ACIDS AND THEIR SALTS.

BY

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(COMMUNICATED BY DR. MITCHELL BRUCE.)

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IN the treatment of rheumatism we observe five distinct epochs: (1) the antiphlogistic, (2) the alkaline, (3) the blistering, (4) the expectant, (5) the salicylate.

The antiphlogistic treatment in English medicine was inaugurated by Sydenham, and was bold, comprehensive, and decisive. It dominated practice until Dr. Fuller, of St. George's Hospital, recommended the administration of alkalies in such doses as to maintain an alkaline reaction of the urine. The chief rival to this treatment was that of Dr. Herbert Davies, who advocated the application of blisters to all the inflamed joints. Then came as the reaction to these methods the "expectant treatment" of Lebert, which was further supported by Sir William Gull and Dr. Sutton in 1866.

For the next ten years the therapeutics of rheumatism was in a state of chaos, which was terminated by the rise of the salicylate treatment, the history of which is peculiarly interesting.

In 1874 Kolbe obtained a crystalline acid (salicylic) by the combination of the elements of carbolic acid with those of carbonic acid. Attention was directed to this compound by its subsequent employment for antiseptic dressing. Later on the internal administration of this acid, and especially of its salt, sodium salicylate, speedily evinced their antipyretic properties.

When given in large doses they reduced the temperature in enteric fever, phthisis, erysipelas, and other affections of a febrile character. Much was expected from their use as antipyretics, but impartial observers showed that the reduction of the temperature was not attended by any modification of the local morbid process, nor by any lessening of the mortality. There was one noteworthy exception to this unfavorable statement, for it was found that when they were administered internally in acute rheumatism the temperature was speedily reduced, and the pain and swelling of the joints disappeared.

In Great Britain, about the same time, and pursuing an independent line of reasoning, Dr. Maclagan tried salicin in the same disease, and with equally satisfactory results in regard to the fever and the affection of the joints.

The connection of the action of the two remedies was shown by Senator to consist in salicin being split up in the living organism by ferments, first into saligenin and subsequently into salicylic acid.

Gradually yet steadily since 1876 the salicylate treatment of rheumatism has grown into professional favour in all lands. Dr. Fagge's words on this point are striking and suggestive: "When I made trial of these drugs I was for a little while sceptical as to their value. The patients rapidly recovered, but I could not forget that I had sometimes seen the administration of other medicines followed

by results which appeared very striking. But when case after case recovered with scarcely a failure I became satisfied that I had a most potent remedy in my hands, and all further experience has strengthened me in this conviction. The immense majority of practitioners and physicians now, I think, entertain a similar opinion."

Now we come to a crucial point. Granted that salicin is changed by the ferment into salicylic acid, is its action superior to or different in any way from that obtained from artificially prepared salicylic acid and its salt of sodium? All experience shows that in a case of acute rheumatism without complications either method will reduce the temperature and relieve the pain within forty-eight hours. Apart from nausea, which is sometimes occasioned by both preparations, there has been a steadily growing conviction that artificial salicylate of sodium—the salt generally given—is depressing, and further that in certain cases there is cerebral excitement culminating in delirium, the patient, it may be, shouting and struggling to get out of bed.

Any hospital physician trying salicin and artificial salicylate of sodium on two rheumatic cases in the same ward, at the same stage of the fever, and in proportionate doses, will find in a given time the patient treated by the salicylate to be weak, exhausted, and perhaps delirious, while the patient to whom salicin has been administered will only suffer, if at all, from slight deafness. True, if the salicylate treatment is discontinued, the patient becomes rational in a few hours, but what would happen if one were to insist on the continued administration of the drug on the human subject I do not know, although after the experimental investigation on animals to which I shall now allude I cannot fail to guess.

The research on the natural and the artificial salicylates on animals was begun in June last, and the results were published in the 'British Medical Journal' of November 30th, 1889. The experiments showed—

1. That salicin in a dose of thirty grains seems to have

no injurious influence on the health of a rabbit, but that it causes a reduction of the temperature about 1°.

2. That salicylic acid obtained from natural sources has no deleterious effect in 10-grain doses.

3. That salicylate of sodium from the natural salicylic acid is not lethal in 32-grain doses, but causes some prostration and lowering of the temperature.

On the other hand, it was demonstrated that—

1. Artificial salicylic acid in a 10-grain dose caused paralysis of the flexors and death.

2. Artificial salicylate of sodium in 18-grain doses causes not merely paralysis of the hind limbs, but paralysis of the fore-limbs and entire loss of control over muscular movements; and death supervenes in a short time from exhaustion, in some cases being preceded by convulsions.

Further, it was shown that salicylic acid and the salicylates of sodium in ordinary use contained an impurity —probably creasotic acid—five grains of which when injected into a full-grown rabbit caused slow respiration and intense prostration, followed by death.

The article referred to concluded with the remark that "in a further communication we shall state the physiological action of the acid from which the impurity has been removed." I must state frankly here what happened. We obtained from the chemical laboratory of the University of Glasgow an acid from which all impurities had been apparently removed. It had a melting-point of 156° C., and was in fine acicular crystals, corresponding to those described in the British Pharmacopœia. But when we injected five grains of it thrice at intervals of fifteen minutes into a rabbit weighing 2½ lbs. the animal died.

We further tried a sample of the salicylic acid of Schuchardt, of Berlin, guaranteed pure, and the same lethal action was the result of the same doses in a rabbit of similar weight.

The conclusion seemed irresistible that artificial salicylic acid could not be purified so as to stand the physiological

test, and Dr. MacLennan and myself moodily reflected that our efforts of four months had been in vain, and that we had detected an impurity, but could not entirely remove it.

Our attention was shortly afterwards directed to an article published by Mr. John Williams in the 'Pharmaceutical Journal' of June, 1878. In this article Mr. Williams stated that he had detected a foreign acid in samples of artificial salicylic acid, which was much more soluble in water than calcium salicylate. On this fact he based a method of separating it, which consisted in saturating a boiling solution of salicylic acid with calcium carbonate, and causing the salicylate of calcium to crystallize out as completely as possible. Upon acidulating the mother liquor the foreign acid was obtained.

"Regarding the medicinal properties of this acid," Mr. Williams writes, "I can say nothing as yet. It may be, like parabenzoic acid, inert, and would then be only a diluent of salicylic acid, or it may be active as an antiseptic, or it may be mischievous;" and he concludes by stating that "until this foreign acid could be removed, neither salicylic acid nor its salts should be used in medicine."

So far as I am aware Mr. Williams did not follow up his investigations, but he had distinctly proved that salicylic acid could be so purified, and that its appearance then was similar to that of the acid obtained from natural sources. This was the key-note of our further research.

We resolved to go upon the lines laid down by Mr. Williams. We set free the salicylic acid by the action of hydrochloric acid on the calcium salicylate, and we simplified his process by slowly crystallizing the acid from hot solutions three or four times repeated. We were thus able without the aid of alcohol, as Mr. Williams recommended, to produce purified acid resembling the natural acid in its appearance, and in no way differing from it by the test of the melting-point, which is 156° C.

Specimens of these purified crystals, and also of those of salicylic acid from natural sources, I now show to

this meeting, as verifying the statements which have been made.

On three different occasions, December 19th and 20th, 1889, and on January 19th, 1890, we tested the physiological action of these purified specimens. The result was most satisfactory. We gave three injections of five grains each to rabbits weighing 2½ lbs., and we found "there was no paralysis, and no depression, but that on the contrary the animals were neither up nor down, but able to run about with ease after the last injection."

For the purpose of estimating the loss by purification, and the time involved in the process, we weighed out one ounce (480 grains) of the salicylate of sodium, and we found that out of the corresponding 414 grains of salicylic acid, 140 grains were first obtained, but by working up the mother liquor nearly the whole of the apparent loss of 274 grains was recovered. Time, ten days.

We weighed out half an ounce of Schuchardt's salicylic acid, and out of the 240 grains 183 grains were obtained, showing a loss of 57 grains by purification. Time, two days.

These facts show that the process of purification, even on a small scale, is not tedious or difficult, and in all probability it could be done easily and thoroughly on a large scale with less loss of time, and at only a slightly increased cost, as compared with the present method.

It may now be asked, "Can no pure specimens of artificial salicylic acid be purchased?" So far we had found none able to stand the physiological test, except those which had been purified at our laboratory. But on January 1st, 1890, unsolicited, a Berlin firm communicated with me through their agents in London. This letter bears incisively on many practical points. They stated that the great bulk of the artificially prepared salicylates used in pharmacy in this country came from their principals; that they guarantee the superior qualities to be free from cresotonic acid, which they believed in reality to be the lethal property discovered in the specimens under our

observation; that their factory passed no acid which did not show a melting-point of 156° C.; that the products of ordinary purity should not be dispensed in pharmacy; and that for every ten pounds of the salicylate of sodium only one pound of it was asked for as manufactured from the purest crystals."

The letter concluded by proposing to send me specimens of their salicylic preparations for my examination and report.

The proposal contained in this letter I accepted, and specimens were received of—

1. Acidum salicylicum extrafein in krystallnadeln.
2. Salicylic acid chemically pure.
3. Sodii salicylas cryst.

On January 11th we subjected these two acids to the experimental test.

1. Acidum salicylicum extraf. 5 gr. were dissolved in 20 minims of rectified spirits and injected into a rabbit at 11.30 a.m.

At 11.45 a.m. the animal passed urine which gave the characteristic reaction with the tincture of the perchloride of iron. It was noted then that the animal looked somewhat dazed and was very quiet.

At 11.55 a.m. the injection was repeated, and at 12.10 p.m. it looked very dazed. No paralysis.

At 12.25 p.m., injection of 5 gr. repeated. The animal assumed a prone position, with the legs stretched out; in five minutes there supervened absolute general paralysis, and at 1.30 p.m. the breathing became extremely shallow and slow, with slight twitchings about the mouth, but no convulsions. Half an hour afterwards it died in a collapsed condition. Ten days later we repeated this experiment with the same acid of the Berlin firm on the rabbit which had stood the test of our purified acid, and we found that this time it was not fatal, but that it caused marked prostration and slight paralysis, which lasted for two hours. The recovery, though slow, was complete.

2. The chemically pure salicylic acid was injected in the

same doses and at the same intervals as in the previous experiment. After the third injection the rabbit became somewhat prostrate and lay with its legs stretched out, but on being roused it was able to move about, though with some difficulty. Its recovery in this case also was complete but slow.

We were rather surprised that these results followed on the use of acids which so closely resembled in appearance the ordinary artificial variety. We made in consequence a few experiments with them in solutions of different strengths, and we found that *their small crystalline* form depended on the rapidity of their crystallization. When this was slow, the crystals became large and well defined, thus showing they had evidently lost the traces of impurity which they had contained. But in the form in which we received them, though guaranteed pure, they were not innocuous. In one instance death was the result, and in the other two abnormal symptoms appeared, which, however, in time passed away. In the artificial variety, which we had purified so as to resemble the natural, these symptoms were entirely absent.

From these experiments we were forced to the conclusion that a high melting-point is not the only or even the best test of purity. Something more is required, and this is that the artificial crystals should be identical with those of the natural variety. Similarity of crystallization seems to be absolutely essential to secure a uniform and harmless physiological action. Pharmaceutical chemists may not have the means or the time for applying the melting-point test, but they can all observe easily the difference between well-defined crystals like those of strychnine and others which present an appearance like quinine. For internal use in medicine they should demand the large crystalline form of the artificial acid, and from this alone should the salicylate of sodium be prepared.[1]

[1] Since reading this paper the firm in question, Messrs. Schering, have submitted to me samples of purified salicylic acid in the form of white acicular prisms, and I was able to satisfy myself that their physiological action was

We trust that the editors of the 'British Pharmacopœia,' in the new Addendum which they are now preparing, will see their way to act on the suggestions we now venture to give.

1. Under "Characters and Tests of Acidum Salicylicum" we would substitute for "white acicular crystals," "the natural acid, in large crystals resembling those of strychnine, but slightly yellowish in colour. The crystals of the artificial acid are similar in form but smaller and whiter. Both acids should have a melting-point of 156° C."

2. Under "Sodii Salicylas: Characters and Tests," instead of "small colourless or nearly colourless crystalline scales," we should say "in large pearly plates."

3. Other varieties of artificial salicylic acid should be termed Poisons, solely intended for external use.

It is rather apart from the object of this paper to say anything about salicylic acid except so far as it is used in pharmacy, but I wish to draw attention to the fact that, owing to its slight taste and powerful antiseptic properties, it is employed for preserving beer, wine, milk, lime and lemon juice, gum, and other fluids. The French seem to have detected its noxious qualities, and have forbidden its use for preserving articles of food; and the Germans lately have acted in a similar manner, and have made it unlawful to use it in the preparation of beer. True, our propensity for drinking beer is not so marked as that of the Germans, yet our brewers should understand that if the acid must be pure when used in medicine, it should be equally pure when placed in this common and pleasant beverage of everyday use.

similar to that of the acid obtained from natural salicylates, and therefore of such purity as to be equally eligible for medical use. I have myself found this purified salicylate of sodium very efficacious in the treatment of acute rheumatism.—'Lancet,' May 31st, 1890.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 62.)

ON THE SYMPTOMATOLOGY

OF

TOTAL TRANSVERSE LESIONS OF THE SPINAL CORD;

WITH SPECIAL REFERENCE TO THE CONDITION OF THE VARIOUS REFLEXES.

BY

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THE symptomatology of total transverse lesions affecting the spinal cord either in the cervical or in the upper dorsal region is a subject of great interest both for the physiologist and for the physician. The physiologist, by reason of his observations upon certain of the lower animals, seems to have instilled into the minds of clinical observers the notion that when the spinal cord is absolutely cut off from communication with the encephalon the reflexes dependent upon the spinal cord below the point of section will, in the course of a very short time—that is, as soon as the immediate effects of shock resulting from the operation

have subsided—exhibit themselves in an exaggerated manner. This general conclusion has perhaps also found favour because of certain other observations upon man himself tending to show that the brain exerts an inhibitory or restraining influence over the reflex activity of the spinal cord. The latter conclusion under ordinary circumstances is undoubtedly quite true, yet it would not be safe to infer that it would also hold good under such very unnatural conditions as must exist when the spinal cord is absolutely cut off from all influences that, under other circumstances, may be exerted upon it by some portions of the cerebrum or of the cerebellum.

As to the observations made by physiologists upon the spinal reflex actions manifested by some animals in whom the spinal cord has been severed from all connection with the brain, I am quite aware that they are at first sight favorable to the notion that in cases of total transverse lesions of the spinal cord occurring in the human subject the reflexes dependent upon the lower portions of the spinal cord would, to say the least, not be diminished. I know quite well that even purposive acts in response to cutaneous stimuli may be manifested by decapitated frogs—acts so complicated and precise as to have given rise to the notion that the spinal cords of these animals must be the seat of a kind of conscious intelligence, capable of accurately adapting response to stimulus. Again, it may be perfectly true that in rabbits and in dogs, in whom the brain has been severed from the spinal cord, reflex actions of a simpler kind are freely elicited, which could only have been produced under the influence of this severed spinal cord. Still, that is not enough to give us a safe warrant for the conclusion that in cases of total transverse lesions in the spinal cord in the human subject the reflexes would, after shock had subsided, become exaggerated. There can be no doubt that the autonomy of the spinal cord diminishes as we ascend in the vertebrate scale. Many of the powers pertaining to it in lower animals are gradually in part taken on by the more developed encephalic centres possessed by

animals higher in the scale. How complicated are the muscular acts producible by decapitated frogs is now a matter of common knowledge. But experiments which have been made in recent years as to the effects of ablation of the so-called "motor centres" from the cerebral cortex of rabbits, dogs, and monkeys respectively, have also made it abundantly clear that the division between encephalic and spinal functions likewise continues to vary in them very considerably.

Such facts as these are abundantly sufficient to instil into our minds the necessity of exercising great caution before we allow our expectations as to what should occur in man to be guided too exclusively by the results of experiments made upon lower animals. I shall not, therefore, pause to consider what precise amount of warranty has been afforded to us by the experiments of physiologists for the general conclusion which undoubtedly prevails in the minds of medical men, to the effect that in patients suffering from total transverse lesions of the spinal cord the reflexes are, after the effects of shock have subsided, subject to some amount of exaggeration. The question which is of more special importance for us is, what does clinical observation teach us upon the subject?

As long ago as 1882 I expressed a very definite opinion upon this subject, because I had, up to that time, had the opportunity of examining over prolonged periods three typical cases of this kind which had been under my care. In Quain's 'Dictionary of Medicine' I spoke thus concerning the symptomatology of "Complete Transverse Softening involving the mid-dorsal region of the Spinal Cord" (p. 1480):—"The temperature in the axilla usually varies between 98° and 100° F., though with an extension of the pathological process, or towards the close of the disease, it may rise to 101°, 102°, or even higher. Meanwhile the lower extremities themselves are often distinctly cold to the hand, the temperature being in some cases more or less subnormal. It is important to note this, because it might have been supposed that hyperæmia and a

slightly elevated temperature would exist, owing to the vaso-motor nerves of the limbs being paralysed.

"The motor paralysis of the lower extremities is absolute, and the abdominal muscles are also powerless. The feet as the patient lies in bed are extended and often inverted, so that the great toes cross one another. The skin after a time tends to become dry and scurfy. The muscles feel flabby to the hand, but they waste only to a slight extent, and continue week after week to show only a small amount, if any, of diminution in the degree of their irritability to faradic and to galvanic currents.

"The sensibility of the limbs is completely abolished both for tactile and painful impressions, as well as for differences of temperature and tickling. A like abolition of sensibility exists over the trunk up to the level of the 'ensiform area,' whilst above this level the sensibility becomes quite natural. The upper limit of anæsthesia may be quite sharply defined, and in these cases of complete transverse softening there is often no distinct 'girdle sensation.'

"The muscles of the lower extremities may show some slight irritability when they are forcibly tapped, and when the soles of the feet are strongly tickled there may be very slight movements of the toes; but beyond this there is often an entire absence of all reflex movements—there is no ankle-clonus, no knee reflex, and a similar absence of the cremasteric and abdominal reflexes. In the initial stages of the affection, however, and especially when the softening is not completely transverse, all these reflexes may be extremely well marked for a time, though they tend gradually to diminish.

"For the first ten days or a fortnight there is often complete retention of urine, but after this time, when the lumbar region of the cord again becomes capable of manifesting to some extent its centric functions, the initial retention gives place to incontinence of urine. This fluid may be discharged at intervals of two to three hours in small quantities, owing to the occurrence of reflex contrac-

tions of the bladder whenever it attains a certain degree of fulness. The passage of a catheter, however, in these cases will often show that the bladder is never completely emptied, two to four ounces remaining after the reflex contractions. Unless special precautions are taken the urine in these cases speedily becomes ammoniacal, and more or less loaded with mucus.

"The bowels are usually constipated, and relieved only after the administration of aperients or enemata. At these times there is generally incontinence of fæces, the patients having no power of controlling the reflex actions concerned in defæcation when they have once been strongly excited. The actual passage of the motion is, moreover, often unfelt."

Other authorities in this country, however, as well as abroad, are not in accord with me in regard to the condition of the reflexes in such affections. Thus, limiting the references to the principal recent writers in this country on the subject of diseases of the spinal cord, I will briefly refer to the opinions expressed by Drs. Ross, Bramwell, and Gowers, in relation to the points in question.

In the second edition of his work 'On Diseases of the Nervous System,' published in 1883, Dr. Ross speaks (vol. i, p. 162) of "the general law that diminution of cerebral influence, other things being equal, increases the reflex activity of the cord." Again, whilst referring to the "morbid physiology" of Acute Diffused Myelitis, he says (vol. ii, p. 103): "When a portion of the grey substance is separated from its connection with the brain by a myelitis situated higher up the cord, reflex actions become increased." A few pages further on he discusses a variety of this disease as "acute transverse myelitis" and concerning it, which is the condition with which we are now more particularly interested, he gives as symptoms for the disease situated in the "dorso-lumbar region" the following (p. 107):—"The paralysed limbs are rigid; the reflexes, both cutaneous and deep, are exaggerated; and there is a tonic spasm of the sphincters. After a time

the lower extremities become œdematous, and their muscles undergo a diffused atrophy; the bladder becomes paralysed, and the urine ammoniacal; acute bedsores appear over the sacrum and trochanters; intermittent fever supervenes, and the patient dies from marasmus." Then he adds, in reference to the disease when it is situated a little higher up in the cord, "In acute dorsal transverse myelitis, however, there is complete absence of bedsores, the bladder is unaffected, and consequently the septicæmia and marasmus are absent." These latter statements are as much at variance with my experience as are the former; it is difficult, indeed, to believe that Dr. Ross can mean these symptoms to refer to the disease where it involves the whole thickness of the cord in either situation; yet he mentions no such limitation, and gives no other account that I have been able to find of the effects of total transverse softening or myelitis.

In the second edition of Dr. Byrom Bramwell's 'Diseases of the Spinal Cord' (1884), in the enumeration of the symptoms of "Total Transverse Lesions," there occurs the following statement (p. 61):—"The reflexes passing through the inferior segments are exaggerated. With the occurrence of secondary degeneration, the paralysed muscles, supplied by inferior segments, become tense and rigid." From what is said on the following page, also, he appears to think that it is the rule to meet with a "band of hyperæsthesia extending round the trunk in the form of a belt," and also with a girdle sensation, so that the "patient feels as if a tight band were drawn round his body."

Dr. Gowers, in his 'Diseases of the Nervous System' (1886), where speaking of "Acute Transverse Myelitis," says (vol. i, p. 225), "The state of reflex action varies, and depends on the position of the disease, in accordance with the laws already stated. An acute lesion in any part of the cord may cause an initial inhibitory loss of reflex action in the part below, but if the lesion is above the lumbar enlargement reflex action returns in the course of

a few hours. Frequently there is no initial depression. Subsequently the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately each attains a high degree of exaltation. . . . The muscles of the limbs are at first flabby and toneless during the stage of initial depression of reflex action, doubtless from the same influence. This condition soon passes off if the lesion is above the lumbar enlargement, and as reflex action becomes active the muscles regain their tone." (See also loc. cit., pp. 136 and 149.)

Elsewhere, when speaking of the determination of the lower level of the lesion in the spinal cord, Dr. Gowers says (loc. cit., p. 156), "To ascertain it we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralysed region —to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are injured. . . . . The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect." And then he adds, "Excess of superficial reflex action indicates withdrawal of the cerebral controlling influence of the reflex centres, and marked excess of the muscle-reflexes suggests the existence of a descending degeneration in the lateral columns, since it implies impaired function of the lowest part of the pyramidal tracts."

In my work 'Paralyses: Cerebral, Bulbar, and Spinal,' published a few months earlier, I had already called attention to what I considered the untrustworthy nature of the second test referred to by Dr. Gowers as a means for indicating the lower level of damage in cases of total transverse lesions of the spinal cord. Referring to this subject, I there said (p. 538), "In cases where extensive transverse lesions exist, situated higher in the cord than the nerves upon which any of these reflexes depend, such reflexes are commonly supposed to be exaggerated in intensity. This is, however, far from being always the

case." After referring to two other possible sources of fallacy in regard to this test I say, "Again, with a total transverse lesion in the lower cervical region, nearly all reflexes dependent upon lower portions of the cord are abolished rather than exaggerated." This being so, it is clear that under certain conditions the "cerebral controlling influence" may be withdrawn with an effect the very reverse of an exaggeration of reflexes; and, on the other hand, it is equally clear, as we shall see, that under similar conditions the most marked descending degenerations may exist in the lateral columns with a complete absence rather than a "marked excess of muscle-reflexes."

I will now give pretty full details concerning four cases of total transverse softening of the spinal cord which have come under my care at University College Hospital during the last eleven years. They are in no sense picked cases. They are, in fact, the only cases in which the lesion has been completely transverse, and where this fact has been verified by an autopsy. In all but one of these cases (No. 3) it was perfectly obvious that the softening through a certain limited part of the lower cervical or of the upper dorsal region of the cord was a total transverse one, seeing that the cord substance thereat was completely diffluent throughout its whole thickness. All the spinal cords were carefully examined after they had been hardened in a solution of bichromate of ammonia. In case No. 3 it was found that both ascending and descending secondary degenerations were just as fully developed as they were in either of the others; and seeing that the symptoms during life were almost precisely similar, it seems safe to conclude that the wider extent though lesser degree of softening which had here existed in the mid-dorsal region had almost sufficed to cut off all encephalic communications with the lower dorsal and lumbar regions of the spinal cord.

Case 1.—Mary F—, æt. 38, a nurse, unmarried, was admitted into University College Hospital under my care April 20th, 1881.

Family history.—Nothing of significance could be ascertained in regard to this.

Past personal history.—The patient had been a governess for twelve years, but six years ago she had to give up this work owing to ill-health. Since this time her occupation has been that of a nurse. She has been very much worried during the last eight years, owing to family troubles. She had always been in good health till six years ago, when she suffered from some nervous complaint, occasioned, as she thinks, by overwork. Under medical treatment she was greatly relieved, and her health continued to improve till May, 1880, when she noticed a lump in her right breast. On account of it she was admitted to this hospital in the following June. She was under Mr. Heath's care, and was treated for "scirrhus of the breast." The breast was removed, and her health greatly improved after the operation. She did not resume her occupation, however, till four months ago. Very soon after this date she began to suffer from great pains in the right shoulder, and soon afterwards in the left shoulder, lasting about half an hour each time: they were very severe at night, and of a lancinating character. The neck and back were subsequently involved in pains of still greater severity; these passed down both arms as far as the elbows: pains were also felt in both thighs, passing from the knees up to the hip-joints. She sought medical advice only three weeks ago, and was soon recommended to come to this hospital.

Present state (April 23rd).—Patient is a rather stout, plethoric woman, lying on her back, and unable to lie on either side on account of pain. She complains of pain in the right breast, in the cervico-dorsal region of the spine, in the shoulders, elbows, and thighs; and of inability to stand or walk. The pains are constant, but paroxysmally worse, and sometimes they are so severe that she shivers.

The skin is everywhere florid; temperature varies from 99° to 98°. The right mammary gland has been removed, and in its place is a puckered irregular scar about three inches long. The cicatrix is adherent to the chest wall, and the tissues for some distance around are indurated and hard—evidently infiltrated with new growth. Between the scar and the sternum there is a hard nodule in the skin about the size of a bean, and the skin covering it is red. Above the cicatrix there is a similar nodule in the skin; and over the sternum are three other nodules, each about the size of a pea. The tissues in the axilla are somewhat thickened, and there are one or two hard tender glands under the pectoralis muscle. The scar is tender, and is the seat of more or less persistent stabbing pain. The left breast contains one large, rather hard lump about the size of a small orange; but there is no puckering of skin or retraction of the nipple, and the gland is freely moveable on the pectoral muscle. There is no enlargement of cervical or axillary glands on this side.

Spinal column.—The seventh cervical vertebra is rather prominent. No other unnatural prominence or curvature exists. There is consider-

able tenderness over the lower cervical and upper dorsal region of the spine; and there is also a great deal of pain referred to the same region.

Nervous system.—No head symptoms or evidence of defective function on the side of any of the cranial nerves.

Sensory apparatus.—There is no loss of tactile sensibility, or inability to appreciate the prick of a pin or to distinguish between heat and cold. She complains of more or less constant burning pains in the elbow-joints, shoulders, and hip-joints, and of a stabbing pain starting in the lower cervical and upper dorsal region of the spine, and passing down through the shoulders to the elbows.

Motor apparatus.—Upper limbs: On the right side she can move her shoulder-, elbow-, and wrist-joints perfectly. She can also flex and extend the fingers. Grasp *nil*. Movements on left side similar to those on right side. Grasp 10. Lower limbs: Patient is unable to stand or walk. She can raise both legs from the bed, and flex hip-, knee-, and ankle-joints perfectly but not very powerfully. Flexion of toes perfect. She cannot raise herself into the sitting posture without assistance.

Reflexes.—Plantar reflex normal on both sides. Abdominal, epigastric, and gluteal reflexes not obtained. Slight ankle-clonus on both sides. Knee-jerk exaggerated on both sides. She complains of occasional jerkings of the thighs.

Previously to yesterday she had had no difficulty in micturition, but yesterday she had retention of urine for eighteen hours, though the bladder acted after a hot fomentation to the lower part of the abdomen, and she has since passed urine twice. The bowels have not been opened for seven days.

The examination of the thorax and abdomen revealed nothing very unnatural, with the exception that the respirations were 24 per minute, and that the breathing was chiefly abdominal, very little movement of the chest occurring; whilst the pulse was 112, small, regular, and compressible.

April 26th.—The pain in the cervico-dorsal region of the spine, in the shoulders, and elbows has continued persistently, though paroxysmally worse, since admission. The pain is relieved by morphia gr. $\frac{1}{4}$ administered every four to six hours. She has also been taking six grains of iodide of potassium with an ounce of infusion of calumba three times a day. Diet: Fish or oysters, with ox-tail soup, custard pudding, and wine ℥v.

May 1st.—For the last thirty-four hours she has had retention of urine. A catheter was passed this morning, and 36 oz. of acid urine were drawn off. Bowels have never been opened except by an enema, which is administered daily. She can flex the hip-, knee-, and ankle-joints and toes very feebly; and she can only just raise the legs from the bed. No anæsthesia. No ankle-clonus exists now on either side. Patellar reflex less marked than on admission. Plantar reflex very slight.

Temperature at 7 a.m. 97·8°; at 11 a.m. 99·2°; and at 6 p.m. 98·6°. The iodide of potassium has been increased to 15 grs. in infusion of calumba ʒj, three times a day.

3rd.—There is complete motor paralysis of the lower limbs, which has come on since yesterday. Tactile sensibility is also diminished below the knee on both sides. Retention of urine still continues, so that it has to be drawn off three times a day. The pains continue to be of about the same severity. Temperature at 7 a.m. 98·2°; at 11 a.m. 99·2°; and at 7 p.m. 98·8°. It has not reached 100° since admission.

6th.—Motor paralysis still persists in lower limbs. Tactile sensibility now lost below hip-joints. Abdominal, epigastric, gluteal, and scapular reflexes absent on both sides. Plantar reflex now abolished on both sides. Ankle-clonus also absent. Knee-jerks very slight.

8th.—The patient was placed upon a water-bed yesterday. On the lower part of back on each side of coccyx the skin is red, and there are three dark discoloured bullæ to be seen. No pain or tenderness over the reddened skin. Tactile sensibility lost between the lower dorsal vertebræ and the umbilicus. Motor paralysis of lower extremities continues. Pains as before. Temperature 98·2°. Pulse 120, small, feeble, and compressible. Tongue clean. Retention of urine. Bowels opened by enemata, and she is perfectly conscious of the act.

11th.—The redness of skin over sacrum has not increased, and no new bullæ are seen. Old bullæ same as at first appearance. Pulse 120, extremely feeble and weak. During the last two days the pains have passed down the arms to the palms of the hands. She shivers a great deal, and complains of twitchings in her back and legs, but no jerkings of the legs have ever been noticed by the nurse. Legs feel cold. Iodide of potassium mixture omitted, and one containing ether and tinct. of digitalis with effervescing saline to be taken three times a day.

13th.—Motor power: Complete paralysis of lower extremities. Abdominal muscles somewhat flaccid and apparently paralysed. No movements of elevation or expansion of chest; breathing is entirely diaphragmatic. Considerable weakness of upper limbs. Movements of shoulders limited on both sides, and accompanied by severe pain. Flexion of elbows perfect; extension not farther than an angle of 120°. Movements of wrist perfect. Cannot flex the fingers into the palm or grasp the dynamometer.

19th.—Since last note the pain has been less severe, but of the same character as before. The upper limbs are much weaker; she can only move her arms a few inches from her side. Other movements much as before. She lies with her arms close to her side, her elbows bent at a right angle, the wrists semi-pronated, and the phalangeal joints semi-flexed. The lower limbs, completely paralysed, are extended and flaccid, and the feet are inverted. There is now complete loss of tactile and painful sensations below the xiphoid cartilage and the ninth interspace.

All superficial reflexes, including the plantar, are abolished. There is also no ankle-clonus or knee-jerk to be obtained on either side. Complete retention of urine exists. The catheter is passed three times a day. The urine is acid. She has been unable to retain an enema since last note. Her bowels have been opened twice with castor oil, and on each occasion she has been unconscious of the act and has passed the motions into the bed. Pulse 70—80, much less weak than formerly. Temperature 99°; it has only once since admission reached 100°; it mostly ranges between 97·5° and 99·5°.

21st.—Since last note patient's temperature has varied from 99° to 102·6°. Pulse 90. Respiration 24. No cough; no expectoration; no dulness in front of chest, and breath-sounds as on admission; no râles. Back not examined. Yesterday the urine began to dribble away about four hours after the catheter was passed. Bowels opened by purgatives; she cannot retain an enema. She is unconscious of the passage of fæces and of urine. There is complete loss of tactile and painful sensations as high as the fifth interspace. No loss of tactile sensibility in upper limbs. Complete paralysis of intercostal muscles below the fifth interspace, and of abdominal muscles. All the muscles of the lower limbs contract when tapped with a stethoscope. There is also a slight plantar reflex when the soles of the feet are sharply tapped. No abdominal or epigastric reflex. No ankle-clonus and no knee-jerks. She takes very little food.

28th.—The loss of tactile and painful sensibility reaches as high as the fourth interspace. Temp. 99°. From date of last note up to yesterday it has ranged between 100° and 101·5°. Bladder washed out twice daily with a weak quinine solution.

June 3rd.—There is a considerable amount of dyspnœa this morning. Expiration is short and forcible, and accompanied by bubbling râles in the throat and all over the chest. No retraction of lower part of chest. Pulse very feeble and thready. Ordered a mixture containing ammonia, ether, and digitalis, together with two ounces of brandy in the twenty-four hours.

4th.—Patient is considerably easier this morning. Breathing quieter. Breath-sounds over front high pitched and accompanied by loud bubbling râles. The prick of a pin is not now felt below the level of the third rib. Has required rather more morphia to deaden the burning and other pains from which she suffers. On May 31st her temperature again rose to 101°, and from that time to the present it has ranged between 100° and 102·6°.

12th.—Patient has had considerable difficulty of breathing during the last three days, owing to the amount of mucus which has accumulated in the bronchi. Pains very severe, morphia only controlling them for a very short time, seldom for more than an hour. Temp. 98·8°; since June 5th it has only once risen as high as 100°.

13th.—Difficulty of breathing increased to-day. She was ordered a hypodermic injection of $\frac{1}{10}$ gr. of apomorphia, and this was administered at 4.55 p.m. Previously to injection the pulse was 92. After fifteen minutes, without any feeling of nausea, the pulse fell to 76, and became feeble and irregular. An enema of ʒss brandy in ʒij of beef-tea was given. Pulse 72, and then 80. No sickness at 5.15, so the fauces were touched with the finger, which induced some retching and the expectoration of a little viscid mucus. Some brandy was given by the mouth. Pulse remained at 80, small and occasionally intermitting. At 5.25 pulse fell to 54, was very weak and almost thready; at 5.34 to 58; at 5.40 to 52, very thready, with respiration embarrassed, hands cold and clammy; expression of moribund type. Ether ℥xvi was now injected over chest, and the pulse slowly but progressively recovered. At about 6 o'clock she began to bring up large quantities of frothy mucus (and for an hour or two afterwards she brought up gulps of mucus at intervals—as though from contraction of bronchi; no vomiting). The breathing afterwards improved materially. At 7 p.m. the pulse was good, about 90, and the patient comfortable.

14th.—Considerably easier to-day. Breathing much better. Pain continues about the same, but is rather more readily controlled by morphia.

17th.—This morning at 3 a.m. patient had a fit of dyspnœa, lasting about fifteen minutes. She became dusky in the face; no coughing; breathing short and shallow. At the commencement of the fit she flexed both arms on to the shoulders, the fingers were also flexed, and the head was bent over on to the left shoulder.

19th.—This morning at 5 a.m. patient had a similar attack of dyspnœa, which commenced in exactly the same manner, and lasted about the same time. But for these attacks the patient has been much more comfortable, and her breathing much easier since the apomorphia. The affection of sensibility over the front of the chest is not appreciably altered; the prick of a pin is still not felt below the level of the third rib. But this afternoon patient complained of a sensation of tingling all over the body and legs. The bedsore has increased, and the tissues around are dark. There is a sore also on one heel to-day.

28th.—Patient was easier during the night. At 7.30 she had an injection of morphia. At 8 a.m., when turned over on to right side to have bedsore dressed, she turned pale, became cyanosed, and died quietly a few minutes later. Ether was administered hypodermically without any effect upon the pulse; and the pupils became widely dilated just before death.

Autopsy (six hours after death).—After removal of the vertebral arches nothing unnatural was seen; but on cutting through the spinal cord just below the bulb and reflecting it, a slight angular curvature was found at the level of the fourth or the fifth cervical vertebra, though

involving one vertebra only. In this situation no new growth was seen, but immediately above the angle the substance of the vertebra felt decidedly softer than natural when pressed upon by the finger. After removing the dura mater, which presented nothing unnatural, the *spinal cord*, about two inches from the point of section, and at a site corresponding with the slight projection above mentioned, was found, for a length of three quarters of an inch, to have only about half the width and depth natural to it in this situation. It was here also soft and flaccid, contrasting notably in this respect with the cord substance above and below. Independently of these signs of softening and atrophy there was no abnormal appearance on the anterior surface of the cord, which presented an average amount of vascularity. Its posterior surface showed the same evidence of atrophy at the site above referred to, but no unnatural vascularity above, below, or over the wasted region. On cutting through the cervical segment of the cord above the wasted portion, the grey matter presented a fair amount of vascularity, not in any way excessive. There was a slightly altered tint in the columns of Goll and in portions of the lateral columns, but otherwise nothing unnatural was seen. On cutting through the softened and atrophied portion of the cord, it was here found to be reduced to a semi-fluid pulp. On making sections through the cord for about two inches below this atrophied and softened region there was distinct evidence of central softening involving the grey matter, since when cut across the central portions swell up above the level of the surrounding white columns. Sections through the remaining dorsal and through the lumbar regions of the cord showed no evidence of softening, nor was any other morbid appearance to be recognised except that the grey matter was rather more anæmic than natural.

Heart: Right side of heart thickly covered with fat. Mitral valve slightly thicker and more opaque than natural. Walls of left ventricle slightly paler than natural, and consistence slightly diminished. *Left lung:* No adhesions; about three ounces of yellowish serum in the pleura. Upper lobe on section found to be semisolid and œdematous. Portions of the upper and middle regions of this lobe as well as the lowest portion of the lower lobe were in a state of more or less well-marked collapse. Nearly a quarter of the tissue in this lung was in such a condition. *Right lung:* Adhesions extensive. No fluid in pleura. Large portions of the surface of this organ were emphysematous, but on section it presented a healthy appearance throughout. There was no collapse and no new growth. *Liver* rather smaller than natural and adherent to under surface of diaphragm. In different parts of its substance were found five nodules of white new growth, varying in size from a small bean to a medium-sized chestnut. Otherwise the cut surface of the organ was rather pale, its lobules were indistinctly marked, and its consistence was slightly above par. *Spleen* of medium size. No appearance of new growth either superficially or within.

Cut surface very dark, and consistence rather softer than natural. *Left kidney* extremely congested; cut surface of deep claret colour throughout. Capsule stripped off easily, leaving surface uniformly congested. No new growth. *Right kidney* presented similar characters; congestion just as well marked, but on the surface there was one small patch distinctly paler in colour, which was found to extend for a slight distance into the substance of the organ. (It appeared to be a commencing patch of new growth.) *Stomach and intestines* presented nothing abnormal. *Ovaries:* Both hard and cartilaginous to the touch; not distinctly larger than natural. On section they were found to be both tough, and showed an excess of fibrous tissue in their interior. *Uterus* considerably enlarged; cavity not lengthened; walls thickened and very tough. From posterior part of fundus, on left side, a peduncular growth of the size of a small orange projected. It was nodulated and extremely hard, and on section was seen to consist almost wholly of fibrous tissue. On the right side of the body of the uterus, and completely obscuring its outline, there was a very tough, hard encapsuled growth, about the size of a small orange. The capsule was vascular and about one third of an inch in thickness, and on section the tumour presented similar characters to those of the pedunculated growth above referred to. *Brain and its membranes* showed nothing unnatural. There was no new growth or focal lesion of any kind.

An examination of the spinal cord after it had been hardened in a solution of bichromate of ammonia showed the following lesions:

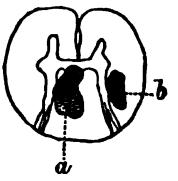
A total transverse softening with greatly diminished bulk of the cord substance existed for a length of about three quarters of an inch, beginning near the middle of the cervical swelling.

A section just above the cervical swelling shows well-marked ascending secondary degenerations in the columns of Goll and in the superficial portions of the lateral tracts.

A section one inch below the lower border of the transverse softening shows descending secondary degenerations in both crossed pyramidal tracts, and to a slight extent in the anterior columns. In addition there are two large, definitely circumscribed, opaque white patches (Fig. 1), the larger of which (*a*) is situated in the central half of the posterior columns, though it does not occupy much of this portion of the column on the right side. The smaller patch (*b*) is oval, and situated just outside the right posterior

cornu. In a section half an inch lower down the latter patch no longer exists, and the one in the posterior columns is smaller and confined to their central region.

FIG. 1.



Half an inch lower still a triangular patch exists in the same situation, which has here broken down into a cavity, and is found to extend downwards for about a quarter of an inch.

Farther down—that is, two and a half inches below the lower level of transverse softening—only descending degenerations appear in the posterior parts of the lateral columns, in the "comma-shaped tracts," and (though very slightly marked) in the inner parts of the anterior columns.

One and a half inches lower—that is, about the mid-dorsal region—descending degenerations are seen in similar situations, being still quite well marked in the "comma-shaped tracts."

In the upper part of the lumbar swelling the descending degenerations are also very well marked in the posterior parts of the lateral columns, and there is a trace of degeneration in the anterior columns, but that of the "comma-shaped tracts" has disappeared.

In the middle of the lumbar swelling the appearances are similar, whilst in the lower third of the lumbar swelling only greatly diminished areas of degeneration in the lateral columns exist.

All through the lumbar swelling, as well as through the lower half of the dorsal region, the grey matter of the cord presents a healthy appearance.

There is no record of an examination of the cervical vertebræ with the view of ascertaining the nature of the disease which led to the softening and projection of a portion of the body of one vertebra, opposite the softened segment of the cord. Nor was the precise pathogenesis of the latter softening quite clear. The projection certainly was not sufficiently great to have caused much pressure upon the cord, though it may have set up primarily an irritation of the anterior columns. At the time of the autopsy there were no appearances in the cord, or in its membranes contiguous to the softened region, in the least indicative of an inflammatory process; nor, on the other hand, was there any evidence of arterial thrombosis.

The pains from which the patient suffered were very severe in the upper extremities, and, strangely enough, severe pains were felt also in the thighs, as well as burning pains in the hip-joints. These latter severe pains are not easily to be explained. From the note of May 19th it would appear that the arms were then in very much the same position as that described by Dr. Thorburn ('Brain,' October, 1888, p. 293) as resulting from disease high enough to paralyse the deltoids, viz. at or just above the level of the fifth cervical nerve. The patient seemed dying from suffocation at the time of the injection of the apomorphia, and it was only ordered in view of the inevitable consequences if the rapidly accumulating mucus could not be expelled. The danger was undoubtedly great from the remedy, but the subsequent relief was no less striking. Strangely enough, too, the great bulk of the mucus was not expelled by vomiting, but rather by what appeared to be successive contractions of the bronchial tubes. From the note made on May 21st it will be seen that the limbs were completely flaccid and paralysed, that all the reflexes (superficial and deep) were absent, but that the idio-muscular contractility was present in all the muscles of the lower extremities when they were tapped with a stethoscope; and possibly what is called in the notes a "slight plantar reflex" was produced in this manner. This idio-

muscular contraction is now generally considered to be quite distinct from a reflex action.[1] According to Schiff, Kühne, and others, it is supposed to be due to the proper excitability of the muscular tissue itself. Funke and Weber have been able to produce the phenomenon in the human corpse even twenty-four hours after death, though Onimus could not elicit it later than eight hours after death.

Case 2.—M. A. W—, æt. 24, a dressmaker, was transferred to my care at University College Hospital on October 14th, 1880, from one of the surgical wards, to which she had been admitted a week previously. She was suffering from recurrent cancer of the left breast with secondary disease of the spine, affecting the spinal cord and causing paraplegia.

The patient's father died of apoplexy, æt. 79, and there is no other history of nervous disease. Her grandmother died of cancer, and her mother, æt. 80, has a tumour of the breast.

Personal history.—The patient is a single woman, there is no specific history, and until a tumour appeared in her breast she never had any illness except "intermittent fever" when seventeen years old. About four years ago she first noticed a tumour in her breast. She was operated upon by Mr. Barker at University College Hospital on April 7th, 1880. When she left she knew that she had some enlarged glands in the left armpit, that the operation had failed in its chief purpose, and that she might expect a return of the growth; and this very soon occurred in the cicatrix and neighbouring skin. About three months after the operation (middle of July) she began to have pains between her shoulder-blades and in both shoulders, though they were neither constant nor very severe. About the same time she also began to have numbness in the inner part of the left wrist and arm, and in the ring and little fingers. This numbness was soon succeeded by severe pain, which kept her "awake for a fortnight." Then it gradually subsided, leaving the whole hand numb. About six weeks ago (beginning of September) she began to feel numbness in the right ring and little fingers and along the inner border of the hand, though to a less marked extent than on the opposite side. About September 24th she began to experience a feeling of numbness in the perinæum, and very soon after in the legs, and then *pari passu* with the increase of the numbness, and equally on both sides, she lost power over her lower limbs. Thus, without any distinct onset or sudden exacerbation, she found increasing difficulty in moving, so that

[1] See "Note sur le contraction idio-musculaire chez les épileptiques," par MM. Ch. Féré et H. Lamy, 'Archives de Physiol.,' Juillet, 1889, p. 570.

by October 1st she was quite unable to move her lower limbs, even in bed. Towards the end of the first week of the paralytic symptoms she had retention of urine, and this persisted for two days after admission to the hospital, so that she suffered from retention for about ten days, and this was succeeded by incontinence. She says also that for about two days before admission the numbness began "to creep up from her waist to her chest."

From the surgical notes it appears that on the patient's admission to the hospital there was a "commencing bedsore on the right side of the sacrum," but this was healed before she was transferred. It is also noted on October 12th that the "patellar tendon-reflex was present and not diminished."

Present state (October 19th).—Some extracts only as to her condition at this time will be given, as the loss of sensibility was far from complete, and therefore at this period a total transverse softening could not have existed.

Although she had lost flesh, she is still a fairly well-nourished woman; and with the exception of the scar and new growth described below there is no affection of the skin, which is warm, moist, and of a healthy colour. The left breast is absent, and in its place is a long scar which extends into the axilla, and ends in a loose fold of skin. The scar is nowhere entirely adherent to the chest. There is much hard thickening of the skin in the neighbourhood of the scar, and also upwards to the summit of the shoulder. The tissues in the axilla are indurated so as to form a hard mass there. Temperature varying between 98° and 99° since admission.

Spine.—There is an unusual prominence of the last one or two cervical and of the first three or four dorsal vertebræ. There is likewise some tenderness to percussion over them, as well as hyperæsthesia to touch, to pin-prick, and to heat and cold.

There is absolute loss of all power of voluntary movement in the lower extremities. There is no wasting of any of the muscles, and the electrical reactions are about normal. In the upper limbs there is no evident wasting of any muscles, except to a slight amount in the ball of the left little finger. Most of the muscles respond rather more readily on the right than on the left side. She can execute any movement with either arm or forearm except flexion of the wrist on the left side, and on that side the power of flexion of the fingers is so much diminished that she cannot close her hand sufficiently to grasp at all. Grasp, right 19, left 0. No action of abdominal muscles either voluntarily or during respiration.

There is greatly diminished but not abolished sensibility, in all its modes, over the whole of the left hand, but especially over the little finger and the palmar and inner side of the ring finger. A similar condition exists on the right side only over the little and ring fingers. Similar defects in sensibility exist over both lower extremities, over the abdomen, and over the thorax as high as the fourth interspaces on both

sides. Above this level sensation to touch, pain, and heat and cold appears to be quite natural.

Reflexes.—The patellar tendon-reflex is absent on both sides. No ankle-clonus usually, though occasionally one may be obtained. Plantar reflex very slight. Abdominal reflexes absent.

Bowels habitually constipated; knows when they are about to act, but has no sensation when fæces are actually passing the anus. Urine is voided in gushes at frequent intervals, unaccompanied by sensation. The urine is alkaline and offensive.

November 3rd.—Patient has been complaining for the last twenty-four hours of a good deal of pain down her left arm. The arm and elbow are œdematous and brawny. Temp. 98·8°, pulse 84, resp. 22. Belladonna and glycerine applied to the arm.

The patient was put upon a water-bed a few days after admission, but in spite of every care a bedsore began to form again about the end of the first week in November. About this period also the bladder was ordered to be washed out twice daily with a weak quinine solution. Up to this time the patient's temperature was mostly normal, only occasionally rising to 99·4°; her appetite also continued good, and she slept fairly well.

December 13th.—The general condition of the patient was carefully re-investigated at this date. From the notes then made I quote the following particulars:

Occasionally she has a little pain in the left thumb, but nowhere else. The left arm is still swollen and œdematous. Her power of moving the upper limbs is decidedly less than it was on admission (especially in the left). On this left side the only movements which remain are some amount of flexion and extension at the elbow-joint (not beyond 90° in one direction or 150° in the other). On right side can raise hand to head and execute all other movements, but has very little power. Grasp practically *nil*; when she attempts to grasp the extensors overcome the flexors. Sensibility in the upper limbs is still only affected in the same area as before, and the degree of impairment is not very notably increased.

Lower limbs.—The skin of the lower extremities is remarkably dry and scurfy. Sensibility is now almost completely lost throughout the whole of both lower limbs. Even when the whole hand grasps the thigh or leg no impression is produced. A pin forcibly driven into the thigh produces no distinct sensation; she thinks "there is something moving, but is not sure." [It was noted at an earlier period that "over the anæsthetic areas a pin-prick draws little or no blood."] Heat and cold produce no impression.

There is still complete inability to move any part of either lower extremity. Reflex action is also entirely abolished in each limb, with the

exception that forcible scratching of the soles of the feet causes slight movement of the corresponding toes. Temp. 98·5°, pulse 62, resp. 18. A bedsore has been forming on the sacrum during the last ten days, which has been dressed with an ointment containing 12 grs. of carbolic acid to an ounce of vaseline, and subsequently with iodide of starch daily.

January 6th, 1881.—The patient was again carefully examined at this date. The condition of the upper extremities was found to be not appreciably different from that recorded above, except that the power of moving the left limb was rather less. It could not be moved at all from the shoulder, and at the elbow there was only a slight power of flexion through 30° (from 60° to 90°). Very slight movement at wrist and metacarpo-phalangeal articulations; none at phalangeal articulations. In the lower limbs, however, sensibility was now completely abolished; and the note made concerning the reflexes was as follows:—"No reflex action on tickling soles of feet, but on tapping soles of feet smartly with a stethoscope, contraction of muscles of front of leg followed. The same thing occurred on tapping the muscles directly. No ankle-clonus. No patellar reflex."

The reflex evacuation of the bladder, however, still continues. The notes say, "Patient passes her urine in gushes about every two hours."

There is now loss of all modes of sensibility on the trunk of the body as high as the fouth interspace; also of movement of abdominal muscles and of intercostals to the same level. The breathing is entirely diaphragmatic.

During the last three weeks the patient has lost flesh considerably, but she has not suffered pain, and has slept without morphia. Her appetite for the last two or three days has been very poor, the tongue being coated with a light fur and rather dry.

Over sacrum the bedsore is deeper and more extensive, and covered with slough. The skin around is red and brawny. On the right heel there is a sore about the size of a shilling (which began about two weeks ago), from which a slough is separating. On left heel there is no sore, but the tissues are indurated. Pulse 68, temp. 99·6°; since December 29th it has risen to 101° or 100° nearly every day.

19th.—Patient has been still losing flesh since last note, and is sinking gradually. The bedsore over sacrum is spreading, and at one part is covered with a black slough. She has complained of more pain again in the left arm, and has had a quarter of a grain of morphia twice a day. Anorexia is extreme. Pulse 76, resp. 26, temp. 101·2°. The urine has been free from albumen throughout. For the last five days she has been taking extra strong beef-tea, and port wine ʒiv daily. The latter is now changed to brandy ʒiv.

26th.—Since last note patient has been complaining of pain in the right arm from shoulder to thumb, similar to the pain on the other side. She is scarcely able to raise this hand to the head. She can move the elbow

and wrist, but is unable to grasp. There is still no loss of tactile sensibility except on the ulnar side of the hand. Patient is sinking; she takes very little food and is very drowsy. She often complains of feeling as if she were choking. Since the 18th inst. her temperature has risen nearly every day to some point between 100° and 101°.

28th.—Patient died last night, sinking very gradually.

Autopsy (14 hours after death).—Whilst opening the spinal canal it was found that at the bottom of the neck and between the shoulders there was a very thick layer of subcutaneous fat, over an inch in some parts, and amongst the fat a whitish new growth. The muscles beneath were not infiltrated in any way, nor were the arches of the vertebræ, though these seemed to be rather unnaturally soft. The posterior and external surface of the spinal dura mater presented a natural appearance throughout; it seemed natural also on transverse section of it (with the spinal cord) just outside the skull. But it was found to be unduly adherent to the bodies of the vertebræ for a length of about five inches in the lower cervical and upper dorsal regions, the adhesion being due to a new growth of whitish colour connecting it with the posterior part of the bodies of the corresponding vertebræ. On opening the dura mater laterally and anteriorly the new growth was found not to have made its appearance on the inner surface of this membrane. The lower two thirds of the cervical swelling of the *Spinal Cord* felt decidedly softer than natural, and opposite its lower extremity (corresponding with the lower margin of adhesion of the dura mater to the bone) the cord showed evidence of extreme wasting; it was here notably flaccid and atrophied, and had an appearance suggestive of pressure, though nothing was found that could have caused pressure. For a distance of about four and a half inches below this point the cord presented a very irregular appearance, owing to the existence of two other areas in which its substance was distinctly softened and atrophied. This was most marked about three and a half inches from the lower end of the cervical swelling, where the cord seems to be even softer and more flaccid than it was above. Immediately below this point, for a distance of about one inch, the cord was also somewhat softer than natural; but below this latter level its consistence seemed to be that of health. On the anterior surface of the cord no large vessels were seen, nor was there any unnatural vascularity; but on examining its posterior surface the cervical and upper dorsal regions were found to be decidedly more hyperæmic than natural. On this posterior aspect of the cord depressions and irregularities were to be seen similar to those found on its anterior surface.

On cutting through the cervical swelling about its middle, the surface of the section seemed to be decidedly softer than natural; whilst in another section through its lower third all parts of the white substance were found to be somewhat diffluent. In some parts here a semi-fluid pulp could be easily scraped from the cut surface, whilst the grey matter

was extremely indistinct on both sides. There was, however, no excessive amount of vascularity.

On making another section just below the cervical swelling, at the region of atrophy with softening, the whole substance of the cord was found to be diffluent throughout: it was represented by a thick yellowish-white fluid. On cutting through the lower atrophied portion, the cord (though softened, flaccid, and atrophied throughout) was found to be not absolutely diffluent.

When sections were made through the cord in the lower dorsal and lumbar regions it was found to be of fairly good consistence throughout. All the sections were, however, decidedly paler than natural, the blood-vessels existing in the grey matter being either smaller or less numerous than usual.

Thorax: On cutting through the integuments it was found that the hardened tissues about the base of the scar corresponded with an infiltrating new growth which has caused distinct adhesion to the ribs. On the inner side of the chest, at about the level of the second and third ribs, the pleura was affected with a whitish growth, and the corresponding portion of the lung was adherent to it; but when torn across the adhesions were found to consist only of fleecy connective tissue. This left pleura contained about twenty ounces of yellowish serum. The lower lobe of the corresponding lung was much compressed and airless, and the lower and posterior parts of the upper lobe were in a very similar state. Only the upper and anterior portions of the upper lobe float in water, other portions sink at once. The right lung presented an old pigmented and puckered patch about three quarters of an inch in diameter at the apex, but there was no other phthisical change, old or recent, nor was there any other notable change about this lung. No trace of cancer exists in either organ. *Heart* of medium size, containing some ante-mortem clots in the right cavities. Mitral valves slightly thicker than natural; aortic valves healthy. Muscular substance of left ventricle paler than natural, and its consistence below par. *Liver* smaller than natural, and some parts of it are unduly tough; no distinct pathological change. *Kidneys* rather small, somewhat congested, and slightly tougher than natural. *Spleen* of medium size, rather firm; on section it is seen to be of a uniform dark colour. *Uterus:* This organ has a small fibroid growth attached to its fundus. Ovaries very thick; fibroid capsules exist, and some whitish cicatricial-like patches within. No distinct evidence of cancer. *Bladder:* This shows no ulceration, but the mucous membrane presents some distinct patches of inflammation. These do not occupy more than one seventh or one eighth of the whole surface of the bladder. *Brain and its membranes* fairly healthy; no cancer found in any part of them.

On sawing vertically through the *spinal column* the bodies of the vertebræ, from the fifth cervical to the second dorsal inclusive, were

found to contain a yellowish-white, firm growth. This occupied the centre of each body, and was surrounded by soft bone. The body of the seventh cervical vertebra had almost entirely disappeared, its place being taken by new growth adherent to the dura mater; part of this was torn away during removal of the cord from the spinal canal. The bodies of the vertebræ above and below were normal, being firm and of pink colour, and contrasting strongly with those that were diseased. The ribs were not infiltrated with new growth, and there was no distinct evidence to show whether or not the growth in the vertebræ had spread by continuity from the axilla, either along the ribs or along the intercostal spaces.

A mass of *lymphatic glands* lying in front of the vertebræ below the pancreas was very considerably enlarged. Externally they had a whitish colour, and on section presented all the appearance of being infiltrated with a cancerous new growth. Chains of glands, less enlarged, also extended downwards along the iliac veins into the pelvis.

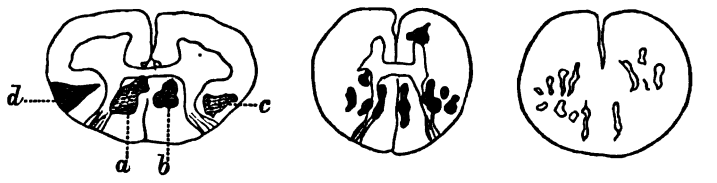
After the spinal cord had remained in a solution of bichromate of ammonia for some time and had become thoroughly hardened, it was again examined, and with the following results:

Throughout the cervical region of the cord well-marked ascending areas of secondary degeneration are seen in the columns of Goll and in the direct cerebellar tracts; but in addition, from about the commencement of the lower third of the cervical swelling up to rather above its middle, there is a continuous longitudinal tract of softening which occupies the central extremity of the left posterior column, together with the hinder part of the grey commissure and the inner part of the posterior grey cornu on the same side (Fig. 2, *a*). This area is rather larger below, and diminishes somewhat in its diameter above. The central portions of the area are now occupied by a cavity, whilst at its periphery degenerated cord substance is seen.

A section through the lower third of the cervical swelling shows, in addition to the area above described, three other morbid patches, irregular in shape but having very much the same sectional area (Fig. 2). One occupies an area on the right side closely corresponding with that already described on the left side of the cord, though with no central solution of continuity (*b*). Another occu-

pies the hinder portion of the right lateral column (*c*); while the third is a wedge-shaped area with its base outwards, and coming to the surface about the middle of the left lateral column (*d*).

FIG. 2. FIG. 3. FIG. 4.



Just below the cervical swelling the cord substance is much wasted, and is diffluent throughout its whole thickness.

For nearly one and a half inches lower down the cord shows no localised lesions except secondary degenerations, though these occupy the greater portion of the white columns of the cord, as both descending and ascending degenerations exist in their most typical form.

The ascending degenerations are due to the fact that a second total transverse softening exists two and a half inches below that in the uppermost dorsal region.

But about one inch above the level of this lower softening the cord begins to show on section, in addition to the above-mentioned areas of secondary degeneration, a number of small opaque white foci (nine in all), agreeing in relative size and distribution with those represented in Fig. 3.

Below the second area of softening for a distance of about one inch the sections of the cord show, in addition to well-marked areas of descending secondary degeneration, a number of very small localised foci of an opaque white colour, very similar in appearance to those which exist above this softened area. In a section three quarters of

an inch below the softening they have such a disposition as is represented in Fig. 4.

In a section three quarters of an inch below that last described no localised areas of change are seen except areas of descending degeneration, and this holds good for all lower portions of the cord—that is to say, for the last four inches of the dorsal region, and for the whole of the lumbar swelling. The secondary degenerations are present in the lateral columns throughout, and in the dorsal region they are also present in the inner part of the anterior columns, but other portions of the white columns and the grey matter show no signs of disease.

This is an interesting case in very many respects. As to the actual cause of the softening nothing very definite can be said. There were no marks of inflammation about the cord; there was no evidence of thrombosis in any of the larger vessels capable of initiating the softening; and there was no evidence of pressure of any kind, although cancer had obviously involved the lower cervical vertebræ and the corresponding outer surface of the dura mater. On October 19th all movements of the upper limbs were possible except flexion of the wrist on the left side. At this date also it will be observed that although the lower extremities were completely paralysed, sensibility was only greatly impaired. Under these circumstances it is important to remark that the reflexes were not altogether abolished. By December 13th the left arm had become greatly swollen and œdematous, and all movements were abolished except slight flexion and extension at the elbow. At this date, though motor power in the legs was completely lost, sensibility to painful impressions was not quite abolished; whilst as to the reflexes it is said, "Reflex action is also entirely abolished in each limb, with the exception that forcible scratching of the soles of the feet causes slight movement of the corresponding toes." By January 6th, however, there was complete sensory as well as motor paralysis of the lower extremities, and now also all the reflexes

were completely abolished; though the notes say that on tapping the soles of the feet smartly with a stethoscope, contraction of the muscles of the front of leg followed—the same thing occurring, however, on tapping the muscles directly. These were doubtless only two different modes of bringing about a simple idio-muscular contraction. Reflex evacuation of the bladder still occurred, the urine escaping "in gushes at intervals of about two hours." Neither in this nor in the previous case is there any mention of the existence of a girdle sensation. The immediate cause of the patient's death here was exhaustion and fever resulting from sloughing bedsores.

Case 3.—H. E—, æt. 51, was admitted into University College Hospital under my care on November 26th, 1884, complaining of loss of power in both legs, but mainly in the right.

Past history.—He has been married twenty-five years; no distinct history of syphilis. He owns to excesses "in drink," especially during his younger days, though they have also occurred occasionally, he says, up to within the early part of this year. In other respects his habits have been regular, and he has always been well fed and clothed. He comes from a healthy and long-lived stock, and there is nothing in his family history to throw light upon his present disease. Till within the last few weeks he has always been a strong, healthy, and well-nourished man, except for two or three slight symptoms referred to below which have been of longer duration.

He says that two years ago, whilst going to his work, he trod on some slippery substance, his right heel slipped, and he fell heavily to the ground. This gave rise to great trembling and faintness at the time. He also says that for a long time, "perhaps two years," he has noticed tremors in the right foot, especially when the heel has been raised, as in resting the toes on a ledge, and that he has for a considerable time complained of pain in the back and a feeling of weakness in the loins.

But the symptoms which have more particularly attracted his attention, and for which he can fix a definite date (viz. the first week in October last), are these:—(1) Bladder troubles; viz. a feeling of tightness about the bladder, and difficulty in expelling his water. (2) The right leg showing a tendency to give way beneath him, and this foot dragging in walking, together with a burning sensation in the right foot and leg—troubles which have since extended to the left limb. (3) Coincidently with these symptoms he began to feel a constant desire to defæcate, with inability to do so; but during the frequent severe straining efforts to micturate (often causing sweating and tremors of the whole body) there

was an occasional involuntary evacuation of fæces. On October 15th, after drinking a pint and a half of ale, he first noticed that his water ran away from him, and it has continued to do so ever since. Previous to his admission he has never been confined to bed; but he has had two rigors, one about five weeks and the other about three weeks since.

Present state.[1]—Patient is a well-built, well-nourished, and fairly healthy-looking man. Temp. 97·6°. He walks with a staggering, uncertain gait, and would soon fall unless supported. He keeps both legs stiff, and brings the heels to the ground first. He raises the right foot with most difficulty. He can sit up and turn over in bed readily. He flexes the knees with little force, especially the right. Movements at all the other joints are more natural. Dynamometer, right hand 72, left 56. Sensibility to touch and pain normal over the whole body, but he confuses impressions of heat and cold all over the lower extremities. Plantar reflexes normal on both sides; cremasteric, abdominal, and epigastric cannot be obtained. Ankle-clonus well marked, and knee-jerk exaggerated on both sides. Patient feels a desire to micturate, but has no control whatever over the act. He always passes his water involuntarily directly he begins to move. He is rather costive, but experiences desires to defæcate, and also has some slight voluntary control over the act. His sexual desires are unimpaired, but erections are rare. There are no tremors or wasting of the muscles, but there is great rigidity in those of the lower extremities, especially on the right side. All muscles respond to faradisation, but those of lower not so readily as those of upper extremities. Patient suffers no pain of any kind; he sleeps well, his memory is good, and he has no unnatural cerebral symptoms. The functions of all the cranial nerves are unimpaired. There are no signs of thoracic or abdominal disease.

December 6th.—Patient on getting out of bed last night found himself unable to stand; his legs were so extremely stiff that he could move his ankles and his knees only to a very slight extent. He also complained of considerable numbness in both legs, but more especially in the right, where the sensation extended as high as the lower part of the abdomen. This morning a soft French catheter was passed, and fifteen ounces of normal acid urine were drawn off. Temp. 98·4°, pulse 68.

7th.—Yesterday after a dose of saline aperient he had an involuntary evacuation of the bowels, of which he was quite unconscious. Neither is he conscious now when his urine passes. This morning he complains of a band-like constriction across the abdomen just below the umbilicus, beneath which level his sensibility is less acute than it is above. His legs are extremely stiff, and tend to get drawn up beneath him—and after this occurs they are with difficulty re-extended. At 2 p.m. a soft catheter was again passed, and 5 oz. of offensive urine were drawn off.

[1] Taken on November 29th.

His temperature, which had previously been normal, soon after this began to rise, so that at 6 p.m. it was 100·8°; at 9.45 it was 101·2°, and at 10.20 it had reached its highest point, viz. 103·6°. The patient then had a rigor, after which the temperature gradually fell, with profuse sweating.

8th.—On examining the patient this morning the knee-jerks and ankle-clonus are found to have disappeared. All rigidity of the legs has likewise disappeared, and they are both now completely paralysed. The pupils, which were equal on admission, have now become unequal, the right being much the smaller of the two. At 3 p.m. the patient shivered, and his temperature, which had dropped to 100°, rose to 102·6° by 5 p.m., though it had again fallen to 99° by 10 p.m. At 7 p.m. a catheter was passed, and a small quantity of urine drawn off, which was rather offensive but distinctly acid. He was ordered an effervescing saline three times a day, and to omit a mixture containing iodide of potassium and liq. arsenicalis, which he had previously been taking. Spoon diet.

9th.—He has slept fairly well, but he passed one or two motions into the bed during the night. Temp. 99·6°. This morning he was placed on a water-bed, and his *present state* was again taken for comparison with that of November 29th.

He has now no voluntary power whatever in his lower extremities, and can neither sit up nor turn over in bed. Movements of upper extremities and of head and neck normal, but the right hand shakes a little when held out. Dynamometer, right hand 65, left 56. Has a feeling as of a constricting band over the abdomen, below the umbilicus, which does not extend to the back. He has complete loss of painful and tactile sensations all over lower extremities and over abdomen as far as margin of thorax in front, and to about the ninth intercostal space in the mid-axillary line. Over the same area he is also dead to thermal impressions. At the upper level there is a narrow zone in which impressions are but faintly appreciated. The upper limit of the anæsthetic area has risen considerably within these last few days. Reflexes: plantar can be obtained on both sides; abdominal, cremasteric, and epigastric not obtainable; ankle-clonus and knee-jerks are now completely abolished. Patient has no control whatever over the sphincters; urine dribbles away at short intervals, and he is unconscious of action either of bladder or rectum. He sleeps well. There is no affection of voice or deglutition; he is not emotional, and does not suffer from delirium, coma, or vertigo. He has, however, slight occasional shooting pains over the vertex. The muscles of the lower limbs are completely relaxed; they are not wasted, and there are no tremors; they respond slightly to the weakest faradic current, and readily to a stronger current. Appetite good, no excessive hunger or thirst, no vomiting, tongue clean and fairly moist.

10th.—Patient slept well last night. Temperature this morning 99°; pulse quiet, 72; plantar reflexes decidedly exaggerated. Right pupil still

smaller than left. Since his legs were tested yesterday with the faradic current they have been the seat of some twitchings.

11th.—This morning some of the urine which had slowly dribbled into a test-glass, and was therefore perfectly fresh, was examined. It was very slightly but distinctly alkaline, smelt offensively, and contained a slight trace of albumen.

15th.—Slept well last night, and is fairly comfortable this morning, with the exception of a constricting pain over the xiphoid cartilage. Temperature is now normal, and since the last note it has ranged between 97° and 99°. He has had no more rigors, but continues to be troubled with the twitchings in his legs. This morning patient was given a glass to collect urine, but it took an hour and twenty-five minutes before any was obtained (showing improvement in bladder reflex); and when it did come it was nearly normal in character, distinctly acid, pale, and with no deposit or offensive odour. Has been taking middle diet for the last two days.

17th.—Patient passed a restless night, only dozing off for slight intervals, and this morning he thought he had a slight rigor soon after 7 a m., but it soon passed off. Temp. 98·4°, pulse 76.

19th.—Patient has not had much sleep during the night, being troubled a good deal with cough. He expectorates with great difficulty. Temp. 100°, pulse 104. Surface temperature of right leg 93°, of left leg 93·6°, and of right arm 97°. Mist. Ammon. c̄ Æth. ʒj, ter die.

22nd.—Patient about the same; cough still very troublesome. Temp. 101·8°, tongue furred, pulse 96. Hot fomentations to chest, together with spoon diet, ordered.

23rd.—About the same. He passed a restless night. Temp. 102·4°, pulse 120, resp. 28.

24th.—Patient is rather worse; still sleeping badly. The urine has again become offensive. A bedsore over the sacrum, which has been forming for the last few days, had to be poulticed last night, and this morning it is beginning to slough. Temp. 101·6°, pulse 120, resp. 48. At 6 p.m. the temperature had risen to 103°, after which it again fell. Brandy two ounces.

26th.—During the last few days patient has been getting much weaker, though he still takes liquid nourishment fairly well. He has also of late been taking Mist. Ammon. c̄ Æth. every three hours. He has not complained of any pain, though he speaks of a girdle sensation (now higher) about the level of the ensiform cartilage. His cough has been very troublesome though not so incessant as it was, and there is great impairment of resonance and bronchophony at both bases. He lies on his back in a semi-apathetic condition, constantly groaning. He does not sleep, bromides and chloral at night producing no effect. The urine has been flowing away at more frequent intervals, and has been ammoniacal for the last two days. The bladder has also during the same time been

washed out with weak Condy's fluid night and morning. Plantar reflex almost abolished on both sides, and both ankle-clonus and knee-jerks still absent on both sides.

At 7 p.m. patient suddenly expired, when being raised to be washed.

Autopsy (fifteen hours after death).—On opening the spinal canal nothing unnatural was seen except that the dura mater in the mid-dorsal region, for a distance of about two inches, was distinctly more vascular than natural, and than it was above and below this level. Large vessels were here seen ramifying over its surface. The anterior surface of the dura mater, however, presented a normal appearance throughout.

When the dura mater was reflected the anterior surface of the *spinal cord* was, perhaps, rather unnaturally pale, from just below the cervical swelling down to about one inch above the lumbar swelling. The vessels throughout this region were rather less numerous than they were either above or below it, except that near the mid-dorsal region there were some enlarged vessels over the right antero-lateral aspect of the cord. Its posterior surface along its whole length seemed rather paler than natural, except for one enlarged vessel filled with coloured clot just below the mid-dorsal region.

The spinal cord for a distance of two inches, beginning a little above the mid-dorsal region, was greatly diminished in consistence in its whole thickness.

On section through this region of maximum softening the cord substance was found to be somewhat pultaceous throughout its whole thickness, though there was no actual diffluence. The section presented an opaque white colour all over, except that the outline of the grey matter could just be detected. No cut vessels could be seen. On section of the cord an inch and a half lower down its substance was found to be still distinctly softer and more flaccid than natural, but the outline of the grey matter was now well defined. Below this, in the lowest part of the dorsal region and throughout the lumbar swelling, sections of the cord presented a normal appearance but for the fact that it looked distinctly anæmic; its consistence was also normal.

When sections were made through the middle of the cervical swelling the grey matter and the antero-lateral columns presented a normal appearance, but the columns of Goll were of a more opaque white colour than natural. A section just below the cervical swelling showed two or three large cut vessels in the central region of grey matter and in the right anterior column; and the column of Goll was altered as above mentioned. A section made two inches and a half below the cervical swelling showed the right anterior cornu to be distinctly more vascular than natural, whilst the outline of the grey matter on the opposite side was not apparent. Another section made about an inch above the commencement of marked softening showed the whole surface to be pale and bloodless and of an opaque white colour, resembling that of the columns

of Goll in the cervical region. A section slightly lower down presented similar appearances except that three enlarged vessels were seen cut across, two in the central end of the left posterior column, and one in the left lateral column.

The *brain and its membranes* presented nothing unnatural save an undue fulness in the vessels of the former, both on its surface and throughout its substance (this congestion being doubtless due to the patient's mode of death).

The great veins and right cavities of the *heart* were much distended with blood, and the right ventricle, in addition, contained a large ante-mortem clot which extended for a short distance into the pulmonary artery. Mitral and aortic valves slightly thicker and more opaque than natural; otherwise nothing unnatural about the heart. *Right lung:* No adhesions and no fluid in pleura. Posterior border and entire lower lobe of lung were deeply congested and more solid than natural. Section of the upper lobe revealed nothing abnormal; but section of the lower lobe showed it to be of a very dark purple colour, semi-solid, and for the most part non-crepitant. A dark reddish fluid exuded from the cut surface, and excised portions of this lobe sunk in water. *Left lung:* Posterior part of left lower lobe covered with recent lymph, slightly uniting pleural surfaces; no old adhesions of any kind. Upper lobe presented no unnatural appearances. Lower lobe was in much the same state as that of opposite side, though it was even more completely solid in many parts, and had about the consistence and friability of splenic tissue. *Liver:* Weight 4 lbs. 4 oz. No thickening of capsule. Its cut surface was almost uniform in appearance and paler than natural. Its substance broke down readily on pressure. *Right kidney:* Weight 7½ oz. It was congested and its capsule stripped off readily, but its substance was slightly tougher than natural. *Left kidney:* Weight 8½ oz.; congested; its capsule stripped off readily, but its upper portion was extremely dense, resisting the firmest pressure of thumb-nail. *Spleen* of medium size; presented nothing unnatural. *Bladder:* Mucous membrane intensely congested; no ulceration.

On examination of the spinal cord after it had been hardened in bichromate of ammonia it was found that for a length of two or three inches, partly above and partly below the mid-dorsal region, diffuse softening existed through the whole of its transverse area, though the different regions were found to be unequally affected in the successive sections that were made. There seemed, in fact, within this longitudinal region of the cord to be a number of small foci of softening affecting different portions of the transverse area of the organ. The effect,

however, upon the cord above and below the region of softening above mentioned was almost the same as if the whole cord had undergone a total transverse softening in some part of the same region—that is to say, in the lower one and a half inches of the dorsal portion of the cord, and in the lumbar swelling, well-marked and typical areas of descending degeneration were found in the lateral and anterior columns; whilst in the upper dorsal region, through the cervical swelling, and on to the bulb equally typical areas of ascending degeneration were found in the columns of Goll, and in the direct cerebellar tracts. But for the presence of these areas of secondary degeneration the cord seemed quite healthy throughout the lowest dorsal and the lumbar regions, as it did above in the upper dorsal and cervical regions.

For about a week after this patient was admitted to the hospital, whilst the motor paralysis of the lower extremities was incomplete, and their sensibility was but little affected, marked ankle-clonus was present, and the knee-jerks were distinctly exaggerated; the plantar reflexes, however, seemed to be about normal, while the cremasteric, abdominal, and epigastric were not to be obtained. By December 9th he had become completely paralysed in both lower extremities; and the marked rigidity which previously existed in them had now given place to complete relaxation. At this date, according to the notes, sensibility was wholly abolished in both lower extremities and over the greater part of the abdomen. As to the reflexes, both ankle-clonus and the knee-jerks were completely abolished, though plantar reflexes could still be obtained on both sides. I am inclined to think, however—judging from what occurred in the other cases as well as from the condition of the cord found after death,—that the abolition of sensibility may not have been quite complete in this case. This view is supported by the fact that on the following day the plantar reflexes had become distinctly exaggerated; whilst the notes also say, "Since his legs were tested yesterday with the faradic current

they have been the seat of some twitchings." And as late as December 15th there is another note to the effect that he was still troubled with "twitchings in his legs." After this he became very ill, with lung symptoms, and no further note was made concerning twitchings, sensibility, or reflexes, except that on the day of his death it is said, "Plantar reflexes almost abolished on both sides, and both ankle-clonus and knee-jerks still absent on both sides." In this case there was a partial girdle sensation encompassing the anterior, but not the posterior part of the body. Although the softening here was extensive in its area it was not carried to the extent of diffluence, and was, in fact, less advanced than in either of the other cases. The cord and its membranes presented no external evidences that the softening was due to an inflammatory process. The death of the patient was brought about in the main by a low hypostatic pneumonia.

Case 4.—Stephen T. H—, æt. 41, a town traveller, was placed under my care in University College Hospital on January 27th, 1880, having been transferred from a surgical ward.

There is nothing of importance in his family history.

Past history.—Patient was formerly an ironmonger, but latterly he has been a town traveller. He married at 24, and his wife has had seven children. He has always been fairly well off, well fed and clothed. He has not had much business anxiety.

Eight years ago patient had smallpox. He has never had rheumatism or scarlet fever. There is no history of syphilis. In June, 1879, he had some pain in his left groin for which he was treated at this hospital, and in the middle of August, whilst stepping into a high gig, he felt a stabbing pain in this groin. At night he discovered a small opening, and a quantity of watery discharge about his dress. He was admitted as an in-patient in October, and left on December 4th. While in the hospital an abscess formed, the size of the palm of the hand, over the lower ribs on the left side, which was opened. When he left the hospital patient says he could walk perfectly well, and had no pains or numbness in any part of his legs.

His present illness began on January 14th. While he was washing himself he suddenly felt his legs give way and he fell down. Both legs failed him, but the left a little more than the right. He was carried to bed, though he afterwards found that he could manage to get out of bed and stumble about for anything he wanted. He could not stand steadily.

He says he had both pains and burning sensations in his legs, especially the left, though he could feel anything touching him as well as ever.

Two days after this attack the sensibility of both legs began to be impaired simultaneously, though the loss was much greater in the left leg than in the right. The numbness in his legs kept getting worse, and crept up from the knee to the thigh. Patient also now found that he could not bear any weight upon his legs, though when he was lying in bed he could kick them about freely.

He was readmitted into the surgical ward on January 24th, 1880. So far as motility was concerned, he was then in the state above described. He could still feel anyone touching his legs, though not distinctly. The following day he passed his motions involuntarily into the bed. He had paralysis of the bladder also, and a catheter was passed. Startings of the left leg occurred first on the day of his admission, and on January 26th startings were also noted in his right leg. On this same day "exaggerated reflexes" were noted on both sides, as well as loss of sensibility up to the ribs. On the following day he was transferred to my care.

His present state was not thoroughly investigated till January 31st, but the following notes were taken.

January 27th.—Patient has only slight power of voluntary motion in the legs, but there are frequent spasmodic twitchings in both limbs, the legs being forcibly flexed at the knees and hips, and also adducted at the hips. There are sudden reflex movements of the legs when the soles of the feet are tickled. The knee-jerks are exaggerated. Ankle-clonus is exceedingly well marked on both sides, though both it and the knee-jerk are rather freer on the right side. There is retention of urine.

29th.—There is absolute paralysis of both legs. There is loss of tactile sensibility in front of both thighs, and on the abdomen as high as midway between the umbilicus and the xiphoid cartilage on the right side and as high as the costal margin on the left side. There is slight sensibility of the soles of the feet, legs, and backs of the thighs. There is no sensation produced by the prick of a pin on the front of the thighs, or over the abdomen in the region above indicated. The prick of a pin is felt, but not as pain, in the feet, legs, and backs of the thighs. These pricks cause reflex movements of the legs. The knee-jerks are absent, and there is no ankle-clonus. Bowels confined, and retention of urine still exists.

30th.—There are spasmodic movements of both legs, though they are much less marked than they were on the 27th. There are no pains or other subjective symptoms. Temp. 101°, pulse 110, resp. 28.

31st. *Present state.*—Patient is a well-nourished, well-developed man, though he says he has been stouter than he is at present. He has no general symptoms of disease. There are no scars or eruptions on the skin except a longitudinal scar running parallel with the ribs, and

situated between the ninth and tenth ribs on the left side. This was caused by an incision made in November last to let out pus. There is also an opening of a sinus between the left thigh and the scrotum, which still discharges an ichorous fluid.

There are no affections of the cerebral nerves or of the upper extremities.

He is absolutely unable to move either leg at any joint. The muscles are fairly well developed, moderately firm, and equally so on the two sides. The muscles of thigh, leg, and special muscles of the foot all react well to both faradic and constant currents, and their reactions are equally good on the two sides. Above the umbilicus there is a slight reaction of both recti to both currents. Below the umbilicus no movements of the recti can be obtained with either current. The oblique muscles react fairly well.

Patient feels the touch of the finger on the left foot, leg, and back of the thigh. Similar results are obtained on the right side. The prick of a pin is not felt on the front of the left foot, leg, or thigh, but on the back of the thigh he sometimes feels it as a touch. On the right side he feels the prick of a pin pretty sharply—in fact, as acutely as in the arm. Except on the dorsum and occasionally on the sole of the right foot, he does not feel either heat or cold, and when he does feel them he mistakes heat for cold.

Over the abdomen the touch of the finger is not felt below the level of the umbilicus; sensation is also impaired between the costal margins and the umbilicus. The prick of a pin is not felt below the umbilicus on the left side. On the right side it is felt as low as midway between the umbilicus and the pubes. Above the umbilicus he can feel the cold spoon applied. A hot spoon he can distinguish as warm. He can feel the cold more distinctly, but is not sure whether it is hot or cold. There are no subjective sensations of numbness or pain except some pain in the right knee and the right shoulder, both of which are also painful on movement. There are occasional starting movements of the legs, but these are slight. The knee-jerk and ankle-clonus are absent on both sides. Tickling the soles of the feet and pricking the legs with a needle, although not felt by the patient, cause starting movements of the legs. Otherwise cutaneous reflexes in the legs and abdomen are absent. Temp. 101·1°, pulse 122, respirations 23.

February 3rd.—The urine is distinctly alkaline. Temp. 102·6° in mouth, 102·6° in left axilla, and 102·4° in left ham.

7th.—The urine now passes very frequently; it does not drop away, but comes in small quantities. Patient still complains of pain in the right shoulder and knee.

8th.—The bowels were opened yesterday several times as the result of an aperient. Patient was not aware when they were going to act. The urine is still voided frequently whenever small quantities collect, but the whole is not expelled, for the catheter passed soon afterwards draws

off two or three ounces. The urine is less ammoniacal than it was, and it does not contain so much mucus. The calf-muscles are flabby; they do not react so energetically to the faradic current as they should do, but reaction to the constant current is about normal.

10th.—The right knee-joint is swollen and contains fluid. It gives no pain. Patient now feels a touch on the dorsum of the right foot and on the front of the right leg, also slightly on the front of the right thigh. On the back of the right leg and thigh he occasionally feels a touch. Over the whole of the left limb the touch is not felt as well as on the right side. He occasionally feels it, but often says he feels when no one is touching him. The prick of a pin is felt on the right side rather acutely over the whole limb. He feels it most acutely on the calf and back of the thigh, and least on the front of the thigh. On the left side the prick of a pin is felt acutely on the front of the leg. It is felt, but not as a prick, on the front of the thigh, but on the back of both leg and thigh he feels it slightly. He feels the prick slightly to midway between the umbilicus and the pubes, though more acutely on the left side. Above this, sensibility is normal. Patient can now move both legs very slightly, the left a little better than the right. On the left side the movement is caused chiefly by flexing the thigh, slightly by flexing the knee. The movement is very slight, and he cannot lift the limb from the bed. On the right side the movement is caused only by flexing the hip. It is just enough to draw the heel up the bed for about two inches. Tickling the soles of the feet causes marked reflex actions in both legs. There is a slight knee-jerk on the left side, but none on the right. There is also no trace of ankle-clonus on the right side, but on the left side a slight quivering of the foot is felt, though it is hardly enough to be seen. The bladder is still washed out daily with a solution of quinine; the urine has now no ammoniacal odour, and its reaction is acid.

14th.—Patient still retains a slight power of movement in the legs; that of the right leg is very slight, whilst that of the left is more marked. Tactile sensibility is now good in both feet and legs, as well as over backs of thighs. There is slight sensibility on the front of the thighs. This is absent or very slight on lower part of abdomen as high as midway between the umbilicus and pubes. Painful impressions are felt acutely on the right leg and thigh, and slightly on the lower part of the abdomen. These are felt badly on the left leg and thigh and lower part of abdomen. Both the legs move when pricked and when the soles of the feet are irritated. There is a slight knee-jerk on both sides to-day; and there is also a slight ankle-clonus on the right side. Micturition is now performed voluntarily. Patient feels the sensation of wanting to pass water, and has time to get the bottle. He can also pass his water voluntarily when only a small quantity has collected. He now knows also when he is going to have a motion, and has some slight control over the act.

15th.—To-day there is a well-marked knee-jerk on both sides, and

ankle-clonus has also returned on both sides. The power of moving the legs is not so good; at the time of the examination the patient could not move them at all. When the catheter (which is a soft india-rubber one) is being passed there are rather violent movements of both legs. There are also spontaneous spasmodic movements of the legs.

17th.—Patient has lost all power of movement in both legs. Sensibility to light touches seems perfect over the whole limb on the right side. Over the left side a touch can be felt, but not so distinctly as on the right side. The slight prick of a pin is felt over the whole of the front of the right leg. He says he feels it more acutely than on the arm. He feels the prick over the whole of the left limb also, but over the front of the thigh it is not felt as pain. Tickling the soles causes well-marked reflex movements of the limbs. Ankle-clonus is well marked, and knee-jerks are exaggerated on both sides. Temp. 99·6°, pulse 120, resp. 18 (taken at 10 a.m., but temp. at 6 p.m. 101°).

March 7th.—Since last note patient's temperature at 6 p.m. has rarely been as low as 100°; it has mostly varied between 100·5° and 102·5°. To-day at the same hour it was 103°. Patient was sick three or four times yesterday, and felt sick all day. There was a considerable flow of pus from the sinus in left groin this morning.

17th.—A small bedsore has appeared on the inner side of the left ankle. Sensibility to tactile impressions is now lost in both lower extremities, and is much impaired over abdomen and chest to the level of nipples. Sensibility to painful impressions is very much impaired over the same area of abdomen and chest, so that as a rule the patient does not feel the prick of a pin. In the lower extremities a deep prick is now and again felt, at other times not, but there is no area to be made out where the sensibility is less impaired than it is at others. The impairment of sensibility is much more marked than it is over the abdomen. There is complete loss of voluntary power over both legs, and very little power in moving the trunk. There is only very slight expansion of the lower part of the chest during inspiration. The knee-jerk is absent on both sides. Ankle-clonus is absent on the right side, but the very slightest quiver of the foot is felt on the left side. The prick of a pin in both legs now and then causes reflex movements. Since the 11th inst. the temperature in the evening has been a trifle under 100°; but to-day at 6 p.m. it was 101°.

April 28th.—The abscess is discharging again after it had ceased for about a week. The temperature during the last fortnight has been higher, several times reaching 102° and 101° in the evening. The pulse has mostly ranged between 120 and 130. Has been taking brandy ʒiv daily for the last week.

May 14th.—The patient suffered from a profuse perspiration which lasted through the night, and he seems much exhausted this morning. Temp. 99·8°, pulse 144, resp. 40.

21st.—Patient has been getting gradually weaker for some days. He

has had some delirium and the pulse has been failing rapidly. The bedsore over the right trochanter (which has existed for more than a month) has become rapidly worse during the last ten days or more; it is now very deep and sloughy. That over the left trochanter is only a little less bad. There is also now a very extensive bedsore over the sacrum exposing the bone. Plantar reflexes, knee-jerks, and ankle-clonus were all absent on both sides two days ago. Patient gradually sank and died at 7 p.m.

Autopsy (sixty-six hours after death).—On opening the spinal canal the left side of the arches of some vertebræ in the lower cervical and mid-dorsal region were, on their inner surface, found to be slightly carious, the erosions being filled up by a soft yellowish-white material. A thin layer of a similar material, looking like half-dried pus, was found also on the corresponding external surface of the spinal dura mater. Otherwise the membranes of the cord presented nothing unnatural.

The *spinal cord* itself was damaged during the opening of the spinal canal in the upper dorsal region. In this region it was found to be reduced to a pultaceous mass, and as this was probably the seat of the main pathological change it was difficult to say how much of the softening was due to damage and how much to disease. The external surface of the cord presented no hyper-vascularity, either anteriorly or posteriorly.

On section through the spinal cord at various parts of the cervical swelling there was no distinct evidence of undue softening in any part. The grey matter possessed its usual amount of vascularity. In the upper part of the dorsal region the cord substance was extremely soft and pultaceous throughout its whole thickness, for a length of more than one inch; whilst above this, as far as the lower end of cervical swelling, the cord seemed rather softer than natural (it might, however, have been due only to the lateness of the autopsy). Sections made through the lower dorsal and the upper lumbar region seemed to show an unnatural amount of softness in the lateral and posterior columns, though it was thought that this also might be a mere post-mortem change owing to the number of hours between death and the autopsy. Sections through the lower part of the lumbar region presented a fairly natural appearance. Viewed externally the lumbar enlargement had a somewhat atrophied appearance.

Brain and its membranes.—The arachnoid was unusually thickened, and generally more opaque than natural. There was also an excess of subarachnoid fluid. The great arteries at the base of the brain were fairly healthy. The brain showed no naked-eye appearances of disease.

Heart.—The pericardium contained 1½ oz. of blood-stained fluid. Mitral and aortic valves rather thicker and more opaque than natural. *Right lung:* This organ was very firmly adherent to the parietes throughout, and there was a thick layer of lymph on the posterior part of the parietal pleura. The upper lobe was more solid than natural, and on section it was found to be very œdematous, and its tissue very

unduly tough. In the lower part of this lobe there was a large patch, about an inch in diameter, thickly studded with miliary tubercles. The lower lobe was somewhat solidified throughout, and more congested and friable than natural, but contained no tubercle. *Left lung:* This was firmly adherent about the apex to the parietal pleura. The upper lobe was unduly hard and semi-solid, and was here and there puckered on its surface. On section at a distance of three inches from the apex and downwards, the lung tissue was studded with a number of discrete and aggregated miliary tubercles, the latter forming indurated patches about three quarters of an inch in diameter. In the lower part of this lobe there were also two small, thick-walled cavities, about half an inch in diameter, like the remains of old abscesses. No tubercle in other parts of this lung; but the lower lobe was somewhat congested and solidified throughout, the tissue being also more friable than natural. *Liver* of medium size, almost uniformly pale everywhere, except for a few areas in which there was some congestion. Consistence below par, and its substance feels greasy. *Spleen* small, very flaccid; cut surface mottled and grumous-looking. Substance distinctly softer than natural. *Kidneys* extremely flaccid and unduly pale. The left organ showed considerable inflammation of the pelvis, with deposits of calcareous matter on some portions of its mucous membrane. The left ureter also showed well-marked inflammation throughout. *Bladder:* It contained a quantity of thick purulent urine. Walls slightly thickened. No ulceration, but radiating away from its neck were lines of slightly inflamed mucous membrane.

Sinuses.—The sinus opening in the left groin just beneath the spermatic cord ran upwards for four inches between the tendons parallel with Poupart's ligament. It was continuous with another sinus running downwards and outwards for about two inches beneath the fascia lata of the left thigh. On cutting through the spermatic cord there was seen, about half an inch from middle line, a sloughy opening, at the bottom of which the probe came into contact with dead bone. Also communicating with this opening there was a sinus which turned round the edge of the adductor longus and there divided into two parts, one running into the adductor magnus, the other arm communicating with a large cavity beneath the adductor longus. This cavity was filled with dirty brown very fœtid pus. It was irregular in form, and lay between the mass of the adductors and the femur. The tip of the small trochanter projected into it, and seemed slightly eroded. A branch of the cavity also passed backwards beneath the neck of the femur, but no "dead bone" could be felt in that situation.

Examination of the spinal cord after it had been hardened in bichromate of potash:

Portions of this cord are unfortunately missing. It

must have been examined at some previous period either by myself or by one of my assistants, and now no notes as to the results of this examination are to be found. I will, however, enumerate the portions of the cord which remain, and state the nature of the changes of which they are the seat.

(1) A part of the upper third of the cervical swelling.

Sections through this part show well-marked ascending degeneration in the columns of Goll and in the lateral columns.

(2) A portion of cord about four inches long from the lower cervical and upper dorsal region, which is much crushed and partly softened.

Sections through the least damaged portions of this show opaque tracts of degenerated tissue occupying in several places the greater part of the transverse area of the cord. On account of the crushing of the cord during its removal, it was impossible to define the extent of the original lesion. From the nature of the lesions above and below, however, in the form of secondary degenerations, it is highly probable that it must have involved the whole thickness of the cord over a certain extent.

(3) The lower dorsal portion of the cord for three inches above the lumbar swelling.

(*a*) Sections through the upper portion of this fragment show well-marked areas of secondary degeneration in the lateral columns posteriorly, but scarcely any in the anterior columns. The substance also looks white and unhealthy in the peripheral third of both posterior columns. The morbid area is ill-defined in outline, and gradually shades away centrally into healthy-looking tissue.

(*b*) A section made an inch and a half lower down presents a very similar appearance. It looks as if there had been slight softening in the peripheral portions of the posterior columns.

(*c*) A section just above the lumbar swelling shows a similar state of the lateral and of the posterior columns. No distinct change is to be seen in the anterior columns,

in the grey matter, or in any other portion of the cord.

(4) The upper portion of the lumbar swelling.

Sections here show some distinct softening of the peripheral third of the posterior columns, in addition to well-marked secondary degenerations in the posterior part of the lateral columns.

(5) Rather more than the lower third of the lumbar swelling.

Sections through this present a healthy appearance, except for very small areas of secondary degeneration in the lateral columns. There is no evidence here of softening of the posterior columns, and the grey matter presents a healthy appearance.

Nothing is known by me as to the cause of the original pain in the groin in June, 1879, and the discharge which first occurred therefrom two months later. The results of the post-mortem examination showed that the opening in the groin was in connection with several very extensive sinuses associated with foul collections of pus and with necrosed bone. It was made probable also that the large abscess that formed over the lower left ribs during his stay in the hospital previous to December, and which was opened at that time, was connected with an offset from the same system of sinuses. Subsequently there came the softening of the spinal cord and the development of tubercle in the lungs, as well as a suppurative caries of the laminæ of some of the cervical and upper dorsal vertebræ. How far either of these processes had to do with the actual development of the softening of the spinal cord is, of course, altogether uncertain. It is worthy of notice, however, that the very abrupt onset of the symptoms of paralysis on June 14th, in the absence of all post-mortem evidence of a hæmorrhage, pointed strongly to these first symptoms being due to a vascular occlusion of some kind. No conditions favouring embolism were met with after death; but it seems just possible that there may have been a thrombosis occurring rather suddenly, and

due, perhaps, to endarteritis set up in connection with some amount of blood-poisoning, consequent upon the unhealthy suppuration in the sinuses. Certainly in this, as in the other cases, there were no external signs of an inflammatory process in connection with the upper dorsal region of the cord or its membranes.

The extent of the original lesion in the upper and mid-dorsal regions of the cord could not be determined, owing to the damage which it had received at the time of removal, as well as to the fact that I could find no record of the previous examination, doubtless made, of certain portions of the organ that were missing. As it happens, this is of comparatively little importance in regard to my main purpose in this paper, because of the fact that a superficial softening of the posterior columns of the cord (the only lesion, apart from descending secondary degenerations, existing in the last three inches of the dorsal region) also extended into the upper third of the lumbar swelling. For though the grey matter in both the upper and the lower parts of the lumbar swelling presented no appearance of disease, this case will not, perhaps, be considered to have the same cogency as either of the others. Yet in some respects it has supplied very important evidence as to concomitant variations between slight degrees of sensibility persisting and slight manifestations of reflex activity. Thus, when the paralysis was incomplete there was rigidity of the lower extremities, together with distinctly exaggerated reflexes; but by January 31st the motor paralysis had become absolute, and sensibility in all its modes had become very greatly impaired, and at this date the knee-jerks and ankle-clonus were absent on both sides, though starting movements of the legs could be induced by pin-pricks on the legs, or by tickling the soles of the feet. Later on, while the motor paralysis was absolute on February 17th, sensibility had very greatly improved. The notes say, "Sensibility to light touches seems perfect over the whole limb. Over the left side a touch can be felt, but not so distinctly as over the right

side;" and with this state of things knee-jerks, ankle-clonus, and plantar reflexes had become well marked on both sides. By March 17th, however, sensibility was again almost completely abolished in both lower extremities; that to tactile impressions was in fact completely lost, but the sensibility to a deep prick was not absolutely destroyed. There was still complete motor paralysis, but in regard to the reflexes the notes say, "The knee-jerk is absent on both sides. Ankle-clonus is absent on the right side, but the very slightest quiver of the foot is to be felt on the left side. The prick of a pin in both legs now and then causes reflex movements." Unfortunately no mention is made as to the plantar reflexes, but this may not unfairly be taken as an indication of their absence; had they been present it would almost certainly have been recorded. Death occurred in the main from exhaustion with bad bedsores after the paralysis had lasted for seventeen weeks. During the course of this illness there were frequent high temperatures, due in part to the unhealthy suppuration going on (together with other changes in the extensive system of sinuses), and partly to the development of tubercle in the lungs.

It has recently come to my knowledge that the statements I had made in 1882, and afterwards in my work on 'Paralyses; Cerebral, Spinal, and Bulbar,' as to the condition of the reflexes in complete transverse lesions, had been received by some with not a little incredulity. It seemed desirable, moreover, that the detailed evidence on which the statements were founded should be published. I accordingly undertook this task.

Three classes of objections, belonging to one or other of the following categories, have been raised by different friends.

(1) It has been said that abolition of the reflexes in total transverse lesions would be a direct consequence of shock, and that if the patient only lived long enough after the establishment of the lesion the reflexes would return;

further, that with the establishment of well-marked secondary degenerations in the lateral columns, more or less of rigidity would supervene in the previously flaccid limbs.

To this objection the cases now recorded (even without the support which will be given by several other cases about to be referred to) seem to me to afford a complete answer. The duration of shock in most cases of disease or injury to the spinal cord may fairly enough be said to vary between a few hours and, at most, a few days. But in the four cases here recorded anything like shock was a very unobtrusive feature, and the patients remained under observation for considerable but variable periods; it was with them a question of weeks rather than of days. Thus, in Case 1, the duration of the paralysis was eight weeks; in Case 2 it was seventeen weeks; in Case 3 it was nineteen days; and in Case 4 it was seventeen weeks. And although there is not in all of the cases a record of the condition of the reflexes up to the termination of the illness, my memory enables me to say most definitely that the limbs in all remained in a condition of flaccid paralysis, with no sign of rigidity, even up to the end.

(2) It has been said, again, that no such abolition of reflexes would occur unless the lumbar region of the cord had been also the seat of damage; some have even seemed inclined to go so far in support of their opinion as to say that the fact of the reflexes continuing to be abolished after the effects of shock had passed off was of itself evidence that the lumbar swelling had also been the seat of some lesion, so sure were they as to the truth of their general principles. A reasoner of this latter type is often hard to be convinced. I venture to think, however, that I have brought forward some valid evidence to show that, at all events in three of my cases, the lumbar swelling was free from disease even though the reflexes were abolished, and all signs of shock had entirely disappeared. This kind of objection is, however, even more completely met by a record of what has happened in certain cases of fracture-dislocation occurring in previously healthy persons

in the cervical or upper dorsal region of the cord, to which I shall presently refer. Here a localised traumatism only has existed in one of these regions, and there has been no reason for supposing the existence of an independent lesion or disease in the lumbar region of the cord.

(3) Lastly, incredulity has been expressed in regard to the truth of my statements, on the alleged grounds that no such flaccidity of limbs and abolition of reflexes had existed in cases either known to or previously under the care of such critics; there had rather been, they said, the very opposite condition of things, viz. rigidity with exaggeration of reflexes. In regard to objections of this order, all I can say is that when these critics have been asked to give me the references to any such cases as they have described, either published or unpublished, the cases to which my attention has been called have, as yet, always proved unsatisfactory—that is, there has been evidence either of a clinical or of a pathological order to show that the cases referred to have not been in reality cases of total transverse lesion. In some of them it has appeared from the notes of such cases that sensibility has only been "impaired," not abolished; whilst in others post-mortem evidence has shown only a partial transverse destruction of the cord at the seat of disease, and the existence of many more or less normal nerve-fibres surrounded by others which have become broken up, or else by a more or less developed overgrowth of connective tissue in the cases that have been of a more chronic order.

The necessity of accepting any such cases as these last referred to, only with the greatest reserve, is shown by the records of a very remarkable case of so-called "compression-myelitis" described by Charcot. The patient referred to died two years after she had been cured of a paraplegia which had been associated with vertebral disease and angular curvature, and the condition revealed by the autopsy is thus described. Charcot says,[1] "The

[1] 'Leçons sur les maladies du système nerveux,' tome ii, 3me ed., p. 93, 1880.

spinal cord in this woman at the level where compression had existed in consequence of Pott's disease was no larger than a goose-quill, and when cut its section was not more than about one third of that of a healthy spinal cord examined in the same region. Its consistence was very firm, and its colour grey; in short, the spinal cord presented all the appearance of the most advanced sclerosis (pl. iii, fig. 1, *d*). . . . . Above and below this narrowed portion the white columns were occupied by grey tracts of secondary degeneration. . . . . Between the appearances presented by the narrowed portion of the cord when examined by the naked eye only, and the phenomena observed during life, there existed, as it seemed, a most striking and singular contradiction. The restoration of functions, as I have said, had been perfect at the time of death, and yet at this time the cord, if we were not to rely wholly upon the information yielded by microscopical examination, was the seat of lesions so profound that it appeared literally interrupted at one point in its course by a cord of sclerosed tissue, in which one would have thought that every trace of nerve-element had disappeared. . . . . Histology, however, shows us that the contradiction is not real. The connective-tissue substitution is here only apparent. In the midst of the very thick and dense tracts of fibrous tissue which gave to this portion of the spinal cord its grey colour and its dense consistence, the microscope showed a pretty large quantity of nerve-tubes provided with their axis-cylinder and their envelope of myeline, and consequently quite regularly and normally constituted. . . . . It was by the intermediation of these nerve-fibres that, during life, the behests of the will and sensory impressions had been conducted."

This case of Charcot's is undoubtedly a very remarkable one, and difficult enough in many ways (as he points out) to understand; but it suffices admirably to show the very great reserve with which cases of this order should be regarded as cases of total transverse lesions when we

are concerned with the strict estimation of the symptomatology of this latter condition. It is clear that we must not, as I was myself originally disposed to do, accept the mere fact of the existence above and below a given lesion of the best developed ascending and descending secondary degenerations as evidence that the lesion in question was a total transverse one. A very little reflection suffices to show the fallacy of this view, and that such well-developed secondary degenerations in the white columns may exist with all degrees of partial destruction of grey matter; nay, it is conceivable, though not likely ever to occur, that such secondary degenerations should exist in their fullest development without any primary disease of the grey matter at all.

Thus it becomes clear that there are two classes of cases more especially in which we may search for the revelation of the true symptomatology attaching to total transverse lesions of the spinal cord. The first of these classes would comprise the cases (*a*) in which a condition of diffluence, or something approaching thereto, has been induced through the whole thickness of the spinal cord in some limited portion of the cervical or upper dorsal region, either by simple thrombotic softening or by actual acute myelitis, provided that in such cases the patient lives sufficiently long after the establishment of the disease. This is the class of cases which I have hitherto considered in this paper. I have, as yet, not been fortunate enough to find a record of any similar cases by other observers, and possibly very few will be found. The first case that occurred in my practice in 1879 struck me very much, and it sufficed thoroughly to rouse my attention in each subsequent case, simply because the phenomena were quite contrary to what I should have expected to occur. I sought, therefore, to obtain good evidence, during life, as to the degree of preservation of the reflexes in conjunction with different degrees of anæsthesia; and, after death, as to the degree of completeness of the lesion. In the clinical examination of these cases

I was aided by a series of excellent observers who were, at the periods referred to, my house-physicians. The observers thus associated with me in the investigation of these cases, and to whom I am indebted for many accurate and painstaking notes, were Mr. Bilton Pollard, Dr. Dawson Williams, Dr. Henry Carr-Maudsley, Dr. William Pasteur, and Dr. J. Walter Carr. I say that possibly few such observations will be found on record because, in the first place, we should be limited to observations that may have been reported during the last ten or twelve years. We could not go back to older records —to periods, that is, when it was not the custom systematically to detail the condition of the various reflexes. Again, for evidence bearing upon this question we should be limited to such cases as lived sufficiently long to enable us fairly to eliminate the possible effects of shock; to cases which were, moreover, fully reported from the point of view of the exact condition of the reflexes in association with different degrees of impairment or abolition of sensibility; and lastly, to cases in which there had been an autopsy and a determination of the question whether the lesion had or had not been one of the total transverse order.

Since the above was written my colleague Dr. Ormerod has kindly brought to my notice one such case of total transverse softening, which occurred last year in the practice of Dr. Gee at St. Bartholomew's Hospital, and in which he had made the autopsy. I have to thank Dr. Gee for permission to use the notes of this case, for an abstract of which I am indebted to Dr. Ormerod.

Case 5.—A. M. F—, æt. 26, a draper's assistant, was admitted on February 26th, 1889. On February 21st he had complained of pain in the middle of the back, with a feeling of constriction around the chest. Two days afterwards he was worse, and on the evening of February 24th his legs became weak, and soon afterwards numb, whilst the paralysis in two to three hours became complete. There was also retention of urine, and constant vomiting, previous to admission.

State on admission.—Paraplegia reaching to about third dorsal nerve;

anæsthesia to the same level. No superficial or deep reflexes. Retention of urine. Bedsore (third day).

[The above is Dr. Gee's "clinical abstract." What follow, Dr. Ormerod tells me, are extracts from the house-physician's notes—Mr. Rivers up to April 1st, afterwards Mr. Symonds, "both very careful and good observers." Dr. Ormerod adds, "The absence of the knee-jerks did, as I know, attract particular attention."]

March 5th.—Incontinence of urine began.

8th.—Patellar reflex present, but very slight. No plantar reflex.

10th, 12th, and 14th.—No knee-jerks obtained. The electrical reactions of the anterior tibial muscles gave the following results:—Faradic irritability fairly good, rather stronger current required than normal; reaction not quite so good as on admission. Some increase of galvanic irritability, but K.C.C. greater than A.C.C., though the difference between them is less than in a normal muscle.

16th.—Knee-jerks present this morning, more in right leg.

19th.—No knee-jerk.

22nd.—Legs jerk a little on being washed.

23rd.—Plantar reflex well marked. *More sensation in legs.*

April 10th.—Reflex movements of legs increased; they often become drawn up when not being touched. *Little or no sensation in legs.* No knee-jerks.

[In reference to the notes concerning sensibility on March 23rd, April 10th, and May 12th, Dr. Ormerod writes, "This appears to refer to sensation of touch; no special note was made as to sense of pain, temperature, &c."]

May 12th.—Involuntary contractions of the legs, causing them to be completely flexed, are more marked; they move at the least irritation to the skin. No knee-jerks. *No sensation in legs.* The anæsthesia now reaches up to the lower border of the sternum. Skin reflexes on abdomen not present.

June 1st.—No knee-jerks.

10th.—Patient died of exhaustion in connection with bedsores and bladder troubles. "The autopsy showed softening of the cord, involving the whole section apparently, the maximum amount being at the level of the third dorsal segment." It extended in length for about two inches. After hardening the cord, Dr. Ormerod says, "The lumbar region was found to be normal except for descending degenerations."

This case affords a valuable confirmation of the truth of my observations. The state of the lower extremities, and their condition as to reflexes and twitching movements, agrees in the closest manner with what was found at different times to exist in my Cases 3 and 4, where it seems certain that the loss of sensibility though nearly

was not absolutely complete. There was the same kind of spontaneous drawing up of the legs, with twitchings when the limbs were touched or pricked. Then, again, some return of tactile sensibility was noted in Dr. Gee's case on March 23rd; and, as Dr. Ormerod points out, no special notes were made as to the patient's insensibility to painful impressions. The notes of my own Case 4, for March 17, however, show that such movements of the legs co-existed with an incompletely abolished sensibility to pain. Yet in my case, as in Dr. Gee's, there was at the autopsy, "apparently" a total transverse lesion. No rigidity of limbs seems to have existed in this case: in reply to my question Dr. Ormerod writes, "No rigidity at first; no mention of rigidity afterwards, unless the 'involuntary contractions' be taken as a form of rigidity." But, as above pointed out, there is reason to believe that these involuntary contractions co-existed with a severance not quite complete of the spinal cord from the brain. Further, in my Cases 1 and 2, in which the severance of the cord from the brain was undoubtedly complete, the limbs were altogether flaccid, and showed neither spontaneous nor reflex contractions of any kind.

The second class of cases in which we may expect to obtain the true symptomatology of total transverse lesions of the spinal cord is represented by (*b*) fracture-dislocations of the vertebræ, associated with complete but limited crushing lesions of the cord, in patients who live long enough for the immediate effects of shock to subside. The following are recorded cases of this type which I have met with, together with three unpublished cases that have been kindly brought to my notice by my colleague, Dr. Tooth.

1. A case recorded by Dr. Tooth ('St. Bartholomew's Hospital Reports,' vol. xxi, 1885, p. 140) of a man who had fallen from a scaffold, resulting in a fracture-dislocation of the fifth and sixth dorsal vertebræ with complete transverse crushing of the cord opposite the former vertebra. "It had the appearance of having been cut across without injury to the membranes." This man lived twenty weeks and four days after the accident.

During life there was total paralysis, with loss of sensibility, in the lower extremities, and in the trunk to the level of the sixth ribs. "No knee-jerk, ankle-clonus, cremasteric or abdominal reflex could be elicited on admission, but the epigastric reflex was present on the right side. Two months after admission it was noted that on pinching the lower part of the thigh the hamstring muscles contracted, but there was no sole reflex. The state of the deep reflexes was unfortunately not noted at this time."[1] Towards the close of his paper Dr. Tooth adds these words: "A curious and hitherto unexplained point in the symptomatology is the complete abolition of all reflexes, superficial and deep, below the lesion shortly after the injury."

2. A case recorded by Kahler and Pick ('Archiv für Psychiatrie,' 1880, p. 297). J. J—, æt. 65, on March 9th, 1878, fell from a height and struck the back of his neck against a beam, whereby he sustained a fracture-dislocation at about the level of the sixth cervical vertebra. His lower extremities and trunk were at once completely paralysed. Sensibility was at first lost to the level of the knees, but the anæsthesia spread upwards during the next two or three days. On examination ten days after the accident (March 19th) his upper extremities were found to be partially paralysed. His breathing was diaphragmatic, and the lower extremities were absolutely paralysed. There was no rigidity; all the muscles were quite flaccid, though not wasted. All modes of sensibility were abolished to the level of the upper part of the thorax. All the reflexes were abolished in the lower extremities except the plantar, which were very weak. The cremasteric and abdominal reflexes were also absent. No subsequent record as to reflexes. Death occurred on the seventeenth day, and at the autopsy the spinal cord was found completely compressed between the sixth and the seventh cervical nerves. There was no damage to the lower parts of the cord, which appeared to be in all respects healthy.

3. Dr. Thorburn's Case 1, recorded in 'Brain,' January, 1887, p. 511. This was also a case of fracture-dislocation, in which there was complete paralysis of all nerves below the fifth cervical, with corresponding loss of motion and sensibility. The man lived for twenty-five days after the accident, and it is said concerning this patient, "Both cutaneous reflexes and tendon reactions were absent throughout." At the autopsy the cord was found to be compressed for a quarter of an inch, and softened for one to two inches above and below, but, it is said, "the rest of the cord was healthy."

4. Dr. Thorburn's Case 3, recorded in 'Brain,' October, 1888, p. 294. This was a case of fracture-dislocation between the fifth and sixth cervical vertebræ, caused by a fall from a waggon. There was absolute paralysis of the legs and trunk, with anæsthesia extending to the level of the third rib in front. He was carefully examined by Dr. Thorburn between

[1] In my cases ankle-clonus and knee-jerks have always disappeared before the sole reflex.

eighteen and twenty hours after the accident and immediate onset of the paralysis, there having been no loss of consciousness at the time. At this time his temperature was normal, the skin was dry and warm, the pulse was 66 and feeble, whilst the respirations were 18 per minute. All signs of shock seem then to have passed off, but the condition of the reflexes was thus noted:—"The knee-jerk and plantar, cremasteric, gluteal, and epigastric reflexes were absent." He was operated upon about twenty-four hours after the injury, and just within a subsequent similar period he died. At the autopsy it was found that "the dura mater was uninjured, but the cord was flattened opposite the seat of injury, and was much contused for about an inch above and below, containing hæmorrhages in its substance and in the central canal; elsewhere its structure was normal."

5. Dr. Thorburn's Case 4, recorded in 'Brain,' October, 1888, p. 296. This man, about 2.30 p.m. on March 25th, 1887, had another man seated upon his shoulders, when he was pushed backwards against a high counter, and his neck was twisted by the man falling from his shoulders. "For the first few minutes he only noticed pain in the back of the neck, but then his legs began to feel weak, and he lay down; within ten minutes the lower limbs were completely paralysed and insensitive, and he found that he could not straighten the left forearm." The temperature at 4 p.m. was 94·2°, at 8 p.m. 97·6°, at midnight (that is, nine and a half hours after the injury) it was 99·2°, near which point it remained for some days. The motor paralysis was complete, but the loss of sensation was not absolute in the lower extremities. Below the third ribs "there was absolute analgesia, but a vague sensation was conveyed by tickling." The above notes were made on the following morning—viz. about twenty hours after the injury; and for the same period there is this record: "There were no superficial reflexes nor tendon reactions." From the eighth to the eleventh day after the injury, it was noted that "sensation in the trunk and lower limbs seemed to improve slightly." No subsequent note was made as to the sensibility, but on the fifteenth day there is a note saying, "We found distinct knee-jerk on the right side, and on the left a slight reaction could be obtained."[1] A few days after this the patient's temperature began to rise, and he soon died from pneumonia. At the autopsy the body of the fifth cervical vertebra was found to be slightly dislocated forwards. Some blood was effused into the spinal canal at this level, and the cord was here also compressed for about one inch, but in other parts of the cord nothing unnatural was found. In this case all shock seemed to have passed off at the time the note was made concerning the reflexes; their return to some extent at a later period, coincidently with some improvement in sensibility, is quite in accordance with my own observations.

[1] It is quite possible, therefore, that some amount of sensibility still persisted.

6. Dr. Thorburn's Case 7, recorded in 'Brain,' October, 1888, p. 305. This man, when intoxicated, had fallen from a gallery ten feet in height. He was examined by Dr. Thorburn nearly twenty-four hours after his admission, when, apparently, symptoms of shock had passed off, seeing that his pulse was 80, and his temperature 99·4°. The lower limbs and trunk were completely paralysed, the respiration being diaphragmatic. The lower limbs were also said to be completely anæsthetic, as well as the trunk to the level of the second rib in front. "Superficial and tendon-reflexes were all absent." On the following morning, some twelve or more hours later, the patient being in a very similar general condition, the following additional note was made concerning the reflexes:—"The plantar, cremasteric, abdominal, and epigastric reflexes, and the tendon reactions at the ankle, knee, wrist, and elbow, were all absent." This patient died on the tenth day, but no further notes were recorded as to the reflexes. At the autopsy the seventh cervical vertebra was found to be displaced forwards, and the "cord was compressed at the level of the first dorsal vertebra, and softened for a short distance above and below the site of compression, its centre being occupied by an effusion of blood reaching as high as the fifth cervical nerve-roots, in the form of a narrow cone."

7. Case of A. P—, æt. 18, admitted into St. Bartholomew's Hospital under the care of Mr. Willett on June 17th, 1886. This young man had fallen from a height, and had sustained a fracture-dislocation of the seventh cervical vertebra (see 'Lancet,' 1887, pt. ii, p. 261; and Tooth, 'On Secondary Degenerations of the Spinal Cord,' 1889, p. 30).

On admission there was some loss of sensation and paresis in the arms, but complete loss of sensation and paralysis below a line drawn round the body about three inches above the nipples. The absolute loss of sensibility is strongly attested by the fact that manipulation of a fracture of the thigh, sustained at the same time as the spinal fracture, gave rise to no signs of pain. There was complete loss of all reflexes below the lesion—knee-jerks and the cremasteric and plantar reflexes having been particularly looked for. This absence of reflexes continued to the end, rather over six months. No trace of rigidity of muscles was observed, and no note was made of the existence of involuntary twitchings. Death occurred on January 26th, 1887, and at the autopsy a total transverse lesion was found between the eighth cervical and the first dorsal nerve-roots (see Tooth, loc. cit., Fig. 7), whilst in the lower portions of the cord only well-marked secondary degenerations existed.[1]

8. Case of E. T—, æt. 42, admitted into St. Bartholomew's Hospital under the care of Mr. Langton on October 17th, 1887. The patient had sustained a fracture-dislocation of the sixth cervical vertebra, and on

[1] For additional details concerning this case, as well as for the notes of the next two cases, which I have received permission from Mr. Langton and Mr. Willett to make use of, I am indebted to the kindness of my colleague, Dr. Tooth.

admission the lower limbs were said to be quite paralysed and anæsthetic. There was also absence of reflexes. On November 7th it was noted that the sole reflexes were well marked, and that the right knee-jerk had returned, but feebly; the left was not tried. Death occurred on November 24th, and in regard to the spinal cord Dr. Tooth says, "On section at the point of injury the cord appeared to be completely crushed, and no fibres could be seen in carmine-stained specimens; Weigert's method was not used." (This is not a very conclusive case, I merely quote it for what it is worth; it at least suffices to show that there was no exaggeration of reflexes.)

9. Case of T. B—, æt. 45, admitted into St. Bartholomew's Hospital under the care of Mr. Willett on August 7th, 1888. This was a case of fracture-dislocation at about the fourth cervical vertebra. There was complete paralysis of the lower extremities, and more or less of the upper extremities. Complete anæsthesia existed below the level of the fifth rib, and there was also considerable affection of sensibility in the arms. There was a total absence of tendon-jerks in the arms and legs. This patient died more than six weeks after admission (on September 25th), but in the notes furnished to me Dr. Tooth says, "Neither sensation, motion, nor reflexes returned. No note is made as to rigidity, but Mr. Bowlby is quite sure that there was none. Owing to the great difficulty in obtaining a post-mortem examination the cord was not all removed. Mr. Bowlby removed it to the level of the fifth cervical vertebra, thinking that that would include the lesion, but on examination the cord showed only descending degenerations. There was therefore no opportunity of examining the crushed spot." The lumbar region was, however, found to be quite healthy except for descending degenerations.

If we were to look at these cases of fracture dislocation alone, there might be reason to fear that in some of them at least the suppression of the reflexes had been entailed by shock. The fact, however, that in other of these cases a similar abolition persisted long after there could have been any reasonable grounds for attributing the phenomena to shock, tends to eliminate our reserve in this direction, as also does the fact that the same abolition persisted week after week in the cases of disease which I have recorded, as well as in Dr. Gee's case, where from first to last there had been no symptoms of shock at all.

Similarly, if we were to look to my cases alone it would be open to the hypercritical—in spite of all appearances to the contrary—to maintain that there might have been lesions more or less minute in the lumbar swelling of the

cord, to which the abolition of the reflexes was really to be ascribed. But such an explanation loses much of any force that it might have possessed when applied to superficially observed cases, and becomes almost wholly invalid when applied to the other series of cases, viz. those in which previously healthy persons become the subjects of a local and purely accidental damage to a part of the spinal cord far removed from the lumbar region.

The two sets of cases, therefore, mutually illustrate one another, and by their combination tend all the more strongly to support my position that in total transverse lesions of the spinal cord we may expect, contrary to previous views, to find that both superficial and deep reflexes will be abolished.

It will be needless for me now to sum up and recapitulate the symptoms of such lesions as they occur in the mid-dorsal region. I have nothing definite to add, and no distinct alterations to make in the account, based upon careful and repeated observations, given in Quain's 'Dictionary of Medicine' in 1882, and which is in part reproduced here on pp. 153—155. I would only call attention to the fact that the plantar reflex, as a rule, disappears after ankle-clonus and the knee-jerk in cases where all three have pre-existed—that it is, in fact, the last of the superficial or deep reflexes to be obtained; and that what is termed "idio-muscular contractility" (p. 167) may be met with even long after the plantar reflex has ceased to be obtainable. Then, again, in regard to the organic reflexes, it seems clear that two of them not unfrequently persist in these cases of total transverse lesion. We have seen, for instance, that in many cases when a certain amount of urine has collected in the bladder, this organ will contract sufficiently to expel its contents in part—the urine thus escaping "in gushes" at intervals of two or three hours. Again, though obstinate constipation is the rule in these cases, and there is no evidence that the mere accumulation of its own proper excreta will, as in the case of the bladder, lead to reflex contractions of the intestinal tube

adequate to bring about even partial expulsion of its contents, yet, when a stronger stimulus is added, in the form of some purgative or large enema, the reflex activity of the intestine becomes adequately roused—it is roused, moreover, under conditions where all cerebral control is lost, so that complete incontinence results so long as the extra stimulus lasts.

Besides its importance as a mere scientific problem in symptomatology, this question as to the persistence or abolition of reflexes in lower parts of the body in cases of total transverse lesions of the spinal cord is also one of great interest and importance in reference to a point in diagnosis, and no less so in regard to the pathogenesis of certain nervous states hard to be explained.

The problem in diagnosis is, as to the means which we possess of ascertaining during life the lower limits of a lesion in the spinal cord, where either it or another higher up has produced, at a particular level, a total transverse destruction of the organ. The conclusion to which we have now arrived, in regard to the conditions under which reflexes are abolished, impose limits upon our powers in this direction not hitherto anticipated. To this question, however, I have already referred (p. 157).

The question of pathogenesis is one which I have elsewhere[1] treated somewhat at length in a section entitled "The causation of contracture, ankle-clonus, and exaggerated knee-jerk; and the extent to which they are dependent upon cerebellar influence."

The first person, I believe, to start the notion that unrestrained cerebellar influence was largely concerned with the production of rigidities and exaggerated reflexes was Dr. Hughlings Jackson. This was done in a very brief communication in the 'Medical Examiner' for April 5th, 1877, though he has since referred to and developed the same doctrine in two or three other communications.[2]

[1] 'Paralyses; Cerebral, Bulbar, and Spinal,' 1886, pp. 216—229.

[2] 'Med. Examiner,' March 28th, 1878; 'Med. Times and Gaz.,' Feb. 12th, 1881.

The difficulties standing in the way of the acceptance of some such hypothesis as this of Dr. Hughlings Jackson were greatly diminished, I venture to think, by my observations as to the abolition of rigidity and exaggerated reflexes in total transverse lesions of the spinal cord. When cerebral motor influence alone is cut off, it is an admitted fact that we soon have to do with conditions of rigidity and greatly exaggerated reflexes in the paralysed parts; but as soon as the remaining connections of the encephalon with the lower half of the spinal cord are completely severed, as they are in total transverse lesions, there is at once an abolition of all rigidity and of the superficial and deep reflexes. What can be the cause of this complete change in the condition of the limbs? Seeing that the cerebral motor influence was previously cut off, it would seem that the abolition of the rigidity and of the reflexes must now have been due to the severance of the influence of some other encephalic motor organ, whose previous unchecked activity was the cause, either indirectly or directly, of the rigidity and exaggerated reflexes. But what other organ of the kind is there—that is, what other motor organ—save the cerebellum? It was under the influence of such considerations, and after a careful comparison of the various hypotheses which have been started to explain these phenomena, that I came to the conclusion that a notion closely akin to that of Dr. Hughlings Jackson was most capable of explaining all the facts. Yet, as I have pointed out (loc. cit., p. 224), my reasons in detail, dependent upon views as to the precise modes of activity of the cerebellum, were rather different from those which he has set forth.

The doctrine that has hitherto found most favour has been that of Bouchard, Charcot, Brissaud, and others. It starts with certain positions which are common to both explanations of exaggerated tendon reactions and rigidity. These are (*a*) that exalted tendon reactions depend upon an exalted condition of "tone" in the muscles concerned; and (*b*) that the rigidities with which exalted tendon re-

actions are often associated are only higher manifestations of similar phenomena, produced in an essentially similar manner.

It is here, however, that the two principal explanations that have been given of these phenomena part company. According to the view of the French school, which has been so widely adopted in this country, the phenomena are held to be immediate consequences of the degenerative changes set up in the "crossed pyramidal tracts" by injuries to these tracts higher up, either in the brain or in the spinal cord itself. The degenerative changes in the terminal portion of these fibres are supposed to cause an irritative over-action in the related great ganglion-cells of the anterior cornua, and thus to lead to an exaggerated condition of "tonus" in the muscles, and the production of the phenomena in question.

Great difficulties formerly stood in the way of explaining many of the facts without the aid of some such views (although grave objections could always be alleged against them); hence the few adherents which the counter explanation of Dr. Hughlings Jackson has hitherto been able to command. Now, however, it seems to me that the new facts established in this paper will be found to be altogether opposed to the fashionable views above cited, and to be just as much in favour of some modification of the doctrine of Hughlings Jackson.

One grave objection which always seemed to me much opposed to the view of Bouchard, Charcot, and others, was the fact that exalted tendon reactions and contracture are to be met with in many cases where there is every reason to believe that no such causative structural changes as the hypothesis assumes exist in the crossed pyramidal tracts,—as, for instance, for a time after attacks of Jacksonian epilepsy;[1] again, in cases where mere temporary pressure is exerted upon the antero-lateral columns of the cord; and lastly, in many functional conditions, hysterical or

[1] See Dr. Hughlings Jackson's paper, "On a Case of Temporary Left Hemiplegia," 'Med. Times and Gaz.,' Feb. 12th, 1881.

other. Now, however, there appears a graver objection still; it is that in cases of total transverse lesions of the cord, as we have seen, the supposed cause exists to its fullest extent, viz. degeneration in the crossed pyramidal tracts, and yet, instead of exalted tendon reactions with rigidity, even after many weeks in some of the cases there is a total absence of reflexes, and a flaccid condition of the limbs. Here, then, as it seems to me, is the death-blow to the hitherto commonly accepted hypothesis.

Now let us look to the other mode of interpretation; let us see what can be said in favour of the view that "tonus" is in the main due to some encephalic influence exerted upon the spinal cord, seeing that the cutting this organ off from all encephalic influence leads to abolition of rigidity and of reflexes. This general position, as I have formerly urged, would seem to be pretty well established by my observations. Further, it seems highly probable that the potent encephalic influence which is thus cut off, in cases of total transverse lesions of the spinal cord, is that of the cerebellum. We cannot immediately, however, come to such a conclusion.

All that we are entitled to infer at once is that the severance of the cord from the brain greatly diminishes, at all events, what is known as "tonus,"—that is, diminishes it to such an extent that phenomena acknowledged to depend upon it can no longer be produced. Of this broad fact two explanations seem possible: thus it might be said (1) that owing to the mere fact of the complete severance of the lower half of the cord from the brain the nervous tension, so to speak, or degree of molecular activity in the grey matter of the severed portion of the cord, is so lowered as to lead to such a diminution of tonus. That is to say, that mere vague and diffused nerve impulses habitually passing between the brain and the spinal cord may be essential to the proper functional activity of the centres contained in the latter; that such impulses may maintain a condition of receptivity with correlative power of reaction, which in the absence of such conditions be-

comes lost. This is a kind of explanation that might suffice to account for the negative phenomena, the mere loss of the reflexes and of rigidity, but it is powerless for the explanation of other related positive phenomena; that is to say, of and by itself it furnishes no explanation whatever of the fact that when the influence of the cerebral hemispheres alone is cut off we have the production of greatly exaggerated tendon reactions with more or less of rigidity or contracture.

It is, therefore, the insufficiency of the first mode of explanation that compels us to seek for another. Now, the other explanation, that which I have previously offered (loc. cit., p. 219), is this: We may suppose (2) "that the condition known as muscular tonus is mainly due to cerebellar influence acting upon and through the spinal centres; then it may well be that the removal of cerebral influence from certain parts of the spinal cord may allow cerebellar influence to reach such parts of the cord much more freely than natural—that is, as Hughlings Jackson would say, we should have to do with an unantagonised, or, as I would rather say, an unrestrained influx of cerebellar energy."[1] Further evidence bearing upon the relative merits of this and of the other hypothesis was offered in the following remarks:—"The fact that such muscular irritability, in patients suffering from slight contracture, is increased if they take strychnia, has been commonly held to prove that this irritability is dependent upon changes or conditions existing within the spinal grey matter alone. But if we bear in mind that the muscular irritability in such cases is similarly exalted by mental activity or excitement, or by the performance of voluntary movements, and that it is often notably diminished by sleep, we may see in these facts reasons for

[1] In addition to the facts already urged in support of such a view, I cited what had occurred in regard to reflexes in a remarkable case of complete thrombosis of the basilar artery, as well as in a case of ingravescent apoplexy. These facts, however, as I now recognise, are of doubtful cogency, because it cannot with certainty be said that the loss of the reflexes might not have been effects due to shock.

believing that the excitability of the cord increases or diminishes with the excitement or the reverse of some encephalic centres, and that an excessive influence of some kind, producing increased tonus in the paralysed muscles, must reach the related ganglion-cells of the spinal cord through other channels than the damaged pyramidal tract."

Another question now presents itself. Supposing the cerebellum does exercise some such influence as I have postulated upon the various centres in the spinal cord, it may naturally be asked, through what channels are we to imagine this influence to be conveyed? There would seem to be only two possible routes; that is, either through the "comma-shaped tracts," which is to my mind very unlikely, or else diffusely through the grey matter itself, in the same sort of way that impressions of pain are conveyed in the reverse direction.

Now, first of all in regard to the "comma-shaped tracts." I mention them because they are the only outgoing tracts at present known in the cord, the functions of which are sufficiently uncertain to make it just possible that they are accustomed to convey cerebellar incitations to the muscles, and because the views of Dr. Hughlings Jackson are based in part upon the supposed existence of some definite outgoing cerebellar channels in the spinal cord. Thus he says ('Medical Examiner,' March 28th, 1878), "The hypothesis starts with the assumption that the spinal centres receive impulses from both the cerebrum and the cerebellum, which impulses in health interfere with one another (inhibit one another)." His meaning is made clearer by what follows: "In other words, loss of cerebral influence on the spinal centre may permit the rigidity, for then the cerebellar influence is no longer interfered with, and, metaphorically speaking, 'flows into the parts deserted by the cerebral influence.' Hence it is better to say 'unantagonised cerebellar influx' than 'increased cerebellar influx.'" For my own part, I cannot believe that the motor cells in the spinal cord are habitually the seat of antagonising activities emanating

from the cerebrum and the cerebellum respectively; and the fact that the fibres of the "comma-shaped tracts" seem to terminate principally in the upper half of the spinal cord, and to disappear before the lumbar region is reached, is also opposed to the possibility that this as yet unallotted tract of outgoing fibres should have any such function.[1]

The only other channel, therefore, along which the slight molecular pulses could habitually pass from the cerebellum to the spinal cord (whose existence I postulate) is through the grey matter. These molecular pulses, whatever else they may do, may be supposed to be instrumental in maintaining the tonus of muscles throughout the body; while in various morbid states the amount of energy flowing along their habitual channels from the cerebellum (especially when the usual restraining influence of the cerebrum is withdrawn) may be very notably increased, so as to lead to rigidities and contractures. The notion that the grey matter is the channel along which these influences emanating from the cerebellum pass, I am not able to support by any more definite evidence than is to be found in the following facts.

We know that with absolute paralysis of the lower extremities, so long as sensibility is intact (as is so often the case in the paralysis associated with Pott's disease), the knee-jerks are greatly exaggerated, ankle-clonus is present, and there is more or less of rigidity with spasmodic twitchings. This condition of things existed also in the early stage of my Case 3. On the other hand, where there is more and more loss of sensibility, including loss of painful as well as of tactile impressions, the clinical picture changes:[2] after a time we gradually lose the

[1] Concerning the topography of the "comma-shaped tract," see Tooth, "On Secondary Degeneration of the Spinal Cord," 1889, p. 37.

[2] What follows does not hold good for the effects of unilateral paralysis with anæsthesia. I have now, for instance, a young woman under my care in the National Hospital, in whom the right arm and leg are completely paralysed, all modes of sensibility being also lost; but the paralysed limbs are more or less rigid, and the knee-jerk is greatly exaggerated.

rigidities, the spontaneous twitchings, and ankle-clonus; while with still graver impairments of sensibility the knee-jerks and the reflex movements of the limbs when the muscles are pricked may also disappear; or, finally, these last may continue, together with some slight amount of plantar reflex, so long as even a slight amount of sensibility to painful impressions persists.[1] This last condition was seen in my Case 2 on December 13th, and in Case 4 on March 17th. But we know that painful impressions are likewise conducted through the grey matter of the cord. Thus it would seem that the preservation of even the smallest bridge of grey matter may permit some preservation of painful impressions, and may at the same time permit the passage of cerebellar energy in the reverse direction. I have found, moreover, a remarkable case recorded by Dr. Thorburn,[2] some details of which are subjoined, and which bears in a very interesting manner upon this question as to the channel by which the encephalic influence that serves to maintain tonus in the muscles is conducted.

10. J. B—, æt. 34, was admitted into hospital on December 30th, 1885. He was a carter, and whilst loading a waggon a "tippler" full of coal fell upon him, throwing him upon his face, while the coal struck him between the shoulders. On examination several hours after the accident there was absolute paralysis of both lower extremities, with deficient action of the intercostal and anterior abdominal muscles in respiration. Both legs were completely anæsthetic as high as the knees, but thence upwards he had some sensation, although there was distinct numbness as high as a line drawn round the abdomen about two inches below the umbilicus. The plantar reflexes were noted as "almost absent." "On the following day there was still absolute paralysis of the lower limbs, but there was now no anæsthesia. . . . The superficial reflexes and tendon reactions were everywhere absent. . . . The temperature was 98·6° F. in the morning, and 99·8° F. in the evening." On the follow-

[1] In proof of these statements I would refer to what is stated as to Case 3 on p. 178, and in the notes for December 9th; and I would ask the reader to compare what is said on pp. 200 and 201 with the notes made as to Case 1 on May 19th (p. 161) when sensibility was completely abolished.

[2] 'A Contribution to the Surgery of the Spinal Cord,' 1889, p. 48.

ing day the signs of lung troubles with accumulation of mucus became severe, and the day after, January 2nd, 1886, he died. At the *autopsy* the membranes of the cord were seen to be quite normal, as was the external appearance of the cord itself, but "on section there was found to be a dark black hæmorrhage into the central grey matter in the lower cervical and upper dorsal regions. This hæmorrhage, which measured in its vertical extent from 1½ to 2 inches, was in the greater part of its extent situated centrally, occupying the whole of the central grey matter, and extending but little into the white substance, which in its neighbourhood was merely softened and of a faintly yellow tinge. At the lower part, for a very short distance, the hæmorrhage was limited to the anterior cornu of the right side, while the corresponding left horn appeared to be perfectly healthy. Elsewhere the cord was firm, and presented no abnormality."

Now this case seems to have for me almost all the value of a well-devised experiment. On the second day, when all the reflexes were still absent, though, as the notes say, "there was now no anæsthesia," all signs of shock seem to have disappeared. This continued absence of the reflexes with the return of sensibility seems to be distinctly opposed to the teaching of the cases that I have brought forward in this paper. In reality, however, I believe it to be the kind of seeming exception which tends to prove very fully the truth of many of the conclusions at which I have arrived. It tends to show almost conclusively that analgesia is the kind of defective sensibility which is most potential in bringing about a diminution or loss of the reflexes, and therefore the great importance of recording the state of a patient's sensibility to painful as well as to mere tactile impressions; for it can scarcely be doubted that in this case, where the autopsy showed a lesion limited to and invading the whole of the grey matter of the cord for a certain extent, there must have been, though it is not recorded, loss of sensibility to painful impressions. As we have seen, there was here certainly loss or very great diminution of "tonus" in the muscles, seeing that the "superficial reflexes and tendon reactions were everywhere absent."

I would only say a few words in conclusion as to the

functional relations existing between the cerebrum and the cerebellum, and as to the conditions under which an excess of cerebellar influence becomes drafted into the spinal cord.

"In my opinion, the weakening or removal of cerebral influence from the spinal cord leads to the weakening or removal of an inhibitory influence which (operative probably in the pons Varolii) usually regulates or restrains the outflow of cerebellar energy through its median peduncles. I would not in the present state of knowledge attempt to define in what precise way the cerebrum and the cerebellum co-operate with one another in their possible actions upon the different muscles of the body.[1] In the performance of the most automatic actions the cerebellum may come into play to a considerable extent independently of the cerebrum, and such neuro-muscular processes are comparatively little interfered with by unilateral lesions of the cerebrum. In the performance of the least automatic actions, however, the cerebrum takes the lead, and the cerebellum acts only as it is solicited or permitted to act, in directions indicated by the outgoing cerebral incitations. The withdrawal, owing to unilateral lesions, of cerebral influence from muscles which are principally called into action voluntarily is, therefore, well calculated greatly to interfere with 'the balance of power' usually capable of being brought to bear upon such muscles, and may lead, as it seems to do, to their being acted upon in excess by the cerebellum, even when in a state of rest, in consequence of which there is increased tonus, carrying with it exaltation of deep reflexes or even muscular rigidities."[2]

Such effects do not usually manifest themselves to their fullest extent at once; they are immediately increased to some degree, but they go on increasing to an indefinite extent, so that it may be some days before anything like distinct rigidity shows itself. But, as I have said elsewhere,[3] "it may be that in such cases the extra leakage

[1] See 'The Brain as an Organ of Mind,' pp. 503—510.

[2] 'Paralyses; Cerebral, Bulbar, and Spinal,' p. 222.

[3] Loc. cit., p. 225.

of cerebellar energy, which the cerebral lesion permits after the shock occasioned by its occurrence has had time to resolve, has a tendency to go on increasing up to a certain point, because of the gradually lessening resistance (probably in the pons) opposed to any such overflows of cerebellar molecular energy. All nerve actions, whether normal or abnormal, become easier and recur all the more readily the more frequently they are repeated."

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 71.)

ANALYSIS OF 964 CASES

OF

OPERATION FOR CALCULUS IN THE BLADDER

BY

LITHOTOMY AND LITHOTRITY,

WITH REMARKS.

BY

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In the year 1878 I had the honour of presenting to the Society a record of 500 cases of operation for stone in the bladder of the adult male.[1] I now beg to offer a further record of 464, in all 964 cases, and constituting my entire experience from the first case in 1854 to the end of 1889; besides which are four cases of operation for the removal of foreign bodies unassociated with calculus, comprising a total of 968 cases.

Respecting all these I beg leave to repeat a statement made on the occasion referred to, viz. that I possess full

[1] See 'Transactions' Roy. Med. and Chir. Soc., vol. lxi, p. 159.

notes of every one recorded at the time of its occurrence, on a system adopted at the outset and never subsequently changed; while founded on these is a printed catalogue (private) containing the chief particulars of every one, a copy of which accompanies this paper. The name of the medical man who sent me the case or who was present at the operation, for such there almost invariably was, is there given, as well as the after history, often embodying observations extending over several years. Every fact named can be verified by evidence under my hand. I have adopted this plan as satisfactory at all events to myself, desiring before all things to make a clear exposition of my entire experience, having often in past time regretted the want of details relating to that of some skilled and practised operators who have left no numerical statements, and only imperfect records or general impressions of the results they obtained. Whatever I offer here may be accepted as the outcome of my entire work in this department of surgery. Not a single case has been omitted. My object has been to present here an accurate although necessarily very brief study of the data obtained, chiefly in relation to treatment and its consequences; and respecting this it may be permissible to state at the outset that I am not conscious of having entertained undue predilection for any particular method, and have therefore employed the knife and the lithotrite indifferently, according to my judgment, for the requirements of each individual patient.

These 964 calculous cases have occurred in the following proportions in regard of sex and age: in adult males 933, in females 15, in youths and boys 16.

The operations which I have employed are lithotomy by various methods, and lithotrity; and, for a few among the female cases, dilatation and extraction.

1. Lithotrity.—Regarding lithotrity, the first case of which, that of a girl, bears date of 1854, I may say that I adopted at the outset the method of my friend Civiale, then in vogue. At this time the sittings for a stone of

moderate size were short and numerous, and generally without anæsthesia; the *débris* being permitted to issue for the most part by the natural act of micturition, assisted occasionally by washing out the bladder with a syringe through a large silver catheter. For the first seven years I employed his instruments, which were much superior to those then used here, having learned to do so during two or three visits to him at Paris for the purpose, circumstances which led to a very friendly intercourse terminated only by his death. Previously to that event, however, I had designed the first lithotrite with a cylindrical handle, an idea which Messrs. Weiss and Son carried out for me;[1] and Civiale himself during the last year or two of his life approved and employed my new instruments, made for him at his own request by that firm.

It was early in 1865 that Mr. Clover designed and carried out his idea of removing the *débris* produced by the lithotrite by means of an exhausting india-rubber bottle and silver evacuating catheter. I used it for the first time in April, 1865, for a patient (Case 51 in the catalogue) whom I saw with my friend Mr. C. A. Aikin, Hyde Park, and I continued to do so more or less for about twelve or thirteen years. As my experience increased I employed it more freely than at first, and thus diminished materially the number of sittings before considered necessary. Hence the value of an anæsthetic became obvious, and I always advised it when the "bottle," or, as it was subsequently termed, the "aspirator," was employed, since the action was more painful to the patient than that of the lithotrite. After 1872 I rarely operated without it, and therefore preferred the aid of chloroform, which was invariably administered by Clover. But previously to the last-named date I was in the habit, whenever severe cystitis appeared in a case undergoing lithotrity, of employing an anæsthetic at once, that I might empty the bladder at one sitting; having learned by experience that the best way to treat the cystitis was to remove every

[1] See letter from Messrs. Weiss, 'Lancet,' August 20th, 1864, p. 229.

fragment of calculus, accomplishing this chiefly by means of Clover's aspirator. This principle of procedure I strongly advocated in my lectures here; I also made it the subject of clinical remarks after operating for stone at Hôpital Neckar, in Paris (1876–7), at my friend Dr. Guyon's request, before a large number of students there. I contended that the plan of emptying the bladder at a final sitting under these circumstances constituted a great improvement on the method by baths, demulcents, rest, and waiting for irritation to subside, always employed for cystitis during lithotrity at that time, especially in Paris. But it never occurred to me that this practice would be advisable in every case of calculus, as was, soon after this, to become apparent.

In 1878 Professor Bigelow, of Harvard, U.S., introduced his method by a single sitting, based on the assumption that less injury was sustained by the bladder from prolonged manipulation, provided the whole stone was removed at once, than by the irritation caused through prolonged contact with numerous fragments left therein for several days to await subsequent sittings. I was quite prepared to accept this principle, and, testing it without delay, soon recognised its importance and value. Since that time I have adopted it, with two or three exceptions only, for all those cases to which I considered lithotrity applicable, using, however, the same lithotrites as before, namely, those made on the model designed by myself, with the cylindrical handle, &c. I have made various modifications which experience has suggested from time to time in the apparatus for removing *débris*, arriving finally at the aspirator which I have used during the last few years. Hence, having employed the same instruments for crushing, and the same system for removing the material crushed since 1878 as before that date, I have felt myself unable to adopt a new name to denote the improved method which Professor Bigelow proposed. I have continued to perform "lithotrity," the term originated by the illustrious inventor of the crushing operation, adding, in

order to indicate the essential change made by Bigelow, "*at one sitting*," instead of by several.

In connection with the specimens preserved here it is necessary to point out that in endeavouring to collect the calculous matter removed by lithotrity of the early type, that is by numerous sittings, it was never possible to obtain and preserve the whole of the *débris*. A certain quantity was always lost, the task of collecting having been necessarily confided chiefly to the nurses, not always sufficiently attentive to this part of their duty. Among such, however, the specimens may be taken as representing about three fourths of the calculus in each case. It is advisable, when the *débris* of a stone removed by lithotrity is to be preserved, that it should be first dried, then weighed, and the result recorded. Since adopting the method by a single sitting, which renders the proceeding easy, this has been done in every instance. Accordingly almost every case catalogued here, from No. 503 (1878) to the end of the series, has been so reported.

Each one of these calculi has been placed in a glass cell, and is marked by a number corresponding with that in the catalogue, which indicates the case to which the calculus belongs, with its particulars, so that reference can be readily made from the specimens to the particulars, and *vice versâ*.

There is one feature in the collection of which I have to say a few words. Although it contains many large calculi, including a few of remarkable size, there is a considerable proportion of small ones, when compared with most of the old existing collections, obtained only by lithotomy, brought together as they were before the middle of the present century. When the knife was the only means available to remove the stone, few patients ventured to encounter the risk of operation until after some years of suffering, while the surgeon himself rarely recommended it until the stone had attained certain proportions. But as soon as the great superiority of lithotrity, particularly for cases where the calculus is small, had become

evident, the idea which dominated my practice and my teaching was the extreme importance of discovering the stone in the early stage, since the dangers incurred by the patient with a large stone, either from repeated sittings by lithotrity or from the knife, were thus to be avoided. I lost no opportunity of seeking for the calculus when recently developed, and learned slowly, with surprise, how much more frequently it was to be found in the bladders of elderly men than I had been taught to expect. So far from the stone being more common in children than in adults, according to the universal belief at the period referred to, justified as it was by the records of hospital practice, I was soon in a position to affirm that stone was more common among men of sixty years of age and upwards than at any other period of life. For let it be remembered that all existing records of practice, whether found in museums or reported by the operators themselves, from all sources previous to the middle of the present century, showed that half the total number of operations for calculus occurred in childhood and youth. The truth nevertheless is that a very large majority of calculous cases was then, as now, to be found in persons above fifty years of age; but the fact was then unknown; the calculi were simply overlooked, not being suspected to exist, and one obvious cause of the oversight is to be found in the fact that the early symptoms in elderly subjects are extremely slight—a rule with only few exceptions,—contrasting strongly with the marked and painful symptoms rarely absent in the young.

Thus, the slight irritation scarcely felt by elderly patients unless considerable exercise is taken was naturally attributed to commencing enlargement of the prostate, to undue acidity of the urine, to "irritation consequent on gout," &c. Hence examination of the bladder for calculus had usually been deemed for such slight symptoms unnecessary. But further, at the period referred to, when such cases were examined by an instrument, as sometimes happened, it was obvious, on observing the method usually

followed, that the sounds employed, as well as the method of using them, were only adapted to find large calculi, and that a formation about the size of a bean or an almond could only be struck by the merest chance, and had indeed never been seriously sought for or thought of. Such can only be detected with certainty by light and delicate handling with the small curved or beaked sound, at that time unknown, and of which I availed myself some time after its introduction by Mercier, of Paris. Yet it is manifest that no greater boon could be conferred on the calculous patient than that of finding his stone while it is still small, and I venture to regard the keen pursuit of this object, and its realisation in several hundred cases of elderly men, as one of the most important results illustrated by this collection. Of small uric acid calculi alone, including a few oxalic acid, but not reckoning phosphatic calculi so frequent in age—that is, weighing from twenty grains to a drachm, and occurring among men of advancing age, say from fifty-five to seventy-five years—there are no less than 200 in this collection. The fact that a very large number of patients could thus be freed from calculus almost without risk was one of the highest importance. But there was another result not less valuable which subsequently appeared, namely, that such patients could almost invariably be prevented from forming fresh calculus by adopting dietetic precautions at an early period, before the morbid tendency had become too strongly marked; and this has, I confess, been to me a source of extreme satisfaction. I possess a great number of subsequent records concerning patients on whom I have operated once for uric acid calculus, who, having followed instructions in respect of diet and regimen, have had no return; while, on the other hand, the instances in which a fresh acid formation has taken place have occurred among those who have continued to indulge habits of diet favouring its reappearance, or those in whom such habits have existed for many years, or, lastly, in constitutions tainted by marked hereditary influence.

But at an earlier stage still, calculous matter may not infrequently be detected and removed, while existing only in the form of "gravel" or "concretion." We may often remove these small bodies by the aspirator only, particularly those of uric acid, weighing two or three grains or even larger; or we may occasionally dispose of them by a single crushing of a lithotrite. In connection with this subject I may remark that it has long been customary to employ certain terms to describe these bodies according to their size and importance—the visible crystalline deposits as "sand," and the ovoid or irregularly shaped bodies, like grains of wheat, peas, or small beans, as "gravel" and "concretions."

The object in employing these terms has hitherto been to convey general impressions respecting the small formations, and to reserve the word "stone" for bodies of greater size and importance. Nevertheless all these expressions, including even the last named, are sometimes very loosely employed. No doubt it is difficult, perhaps impossible, to define precisely the limit of their meaning in regard of size and weight. But in order to conform as far as possible to the practice of our predecessors I have invariably refused to recognise as "stone" the small bodies described above, maintaining for them a well-defined class of "gravel" and "concretions;" and especially because the removal of small calculous bodies, now that a formidable operation by the knife is no longer necessary, is a matter of extremely small difficulty and gravity. Hence I have uniformly declined to enter in the series of "stones" removed from adult patients any calculous bodies weighing less than about twenty grains. When a smaller one has been met with I have described it as "gravel" or "concretion."[1] I know that the dis-

[1] Thus, some years ago, I washed out from a patient's bladder some five hundred minute uric acid formations, about the size of a pin's head. The total weight was 2¼ drachms. Smaller quantities I have frequently removed, of which examples are presented here. But it never occurred to me to regard these as instances of "stone in the bladder," or to enter them as cases of operation.

tinction is quite arbitrary, but I contend that any weight, whatever it may be, which is agreed to as marking the limit between "stone" and "gravel" must be equally an arbitrary one. Still it is desirable that a distinction should be drawn, and if possible agreed to, or we may have the washing out of tiny bits of gravel of one or two grains even in the adult individual represented and recorded as an operation for "stone in the bladder!"[1] Taking what I venture to believe may be regarded as a common-sense view of the question, I have adopted the twenty-grain limit for myself. Had I reckoned the removal of uric acid and oxalate of lime formations of the size just named, I should have very largely augmented my number of cases; and still more so had I thus regarded the phosphatic concretions which are so often crushed and removed from the bladder of prostatic patients who have long passed all their urine by catheter. Many persons live, subject to this condition, in tolerable comfort for ten or twelve years or more. Such a one after some months of freedom from pain gradually becomes the subject of calculous symptoms, often severe, due to the presence of a phosphatic concretion, weighing perhaps ten or fifteen grains, too large to wash out, but which a single introduction of the lithotrite suffices to remove. This proceeding may be performed sometimes once or twice a year, and thus, for a single individual, the surgeon may have to repeat the process many times. Had I included all these examples in my series the total number would have reached at least two or three hundred cases more than it now does.

2. Lithotomy.—Regarding the series of operations by lithotomy, I commenced with the ordinary lateral operation for the largest calculi, employing the median for those which five-and-twenty years ago were regarded as just outside the scope of lithotrity. Subsequently I tried the medio-bilateral of Civiale and the bilateral of Dupuytren for the first named, not acquiring any marked

[1] This actually has taken place. See 'Brit. Med. Journ.,' 1887, vol. ii, p. 1376.

preference for any one of these methods. It so happened that during the first fifteen or twenty years of my experience no calculus of very unusual size presented itself. I met with several weighing from one and a half to three ounces, and usually removed them by the lateral operation. The supra-pubic operation I performed for the first time in 1864, not for a calculus, but for a foreign body in the bladder of a young woman in University College Hospital. It was a hair-pin lying across the bladder, tightly impacted in this position and defying any fair attempt to remove it by the urethra. The next occasion was in 1877, for a gentleman whose legs were immoveable and extended as the result of spinal disease. The position for lateral lithotomy being impossible, I performed the supra-pubic in this, as in the preceding case, on a staff, the method adopted at that time for removing a large uric acid calculus (Case 456 in the catalogue).

But in the year 1883 I became acquainted with the modification of this operation made by Petersen, of Kiel, and from my experience of its results in the hands of Guyon, of Paris, and others, I advocated its merits in my lectures at the Royal College of Surgeons in 1884. Immediately afterwards a case of large calculus presented itself, and I performed the new supra-pubic operation, for the first time in this country, in July of that year. The calculus was one of pure cystine, and weighed 2¾ oz., the largest of that product I have ever seen. The patient is now living and well (see Case 690). Several other examples soon came under my notice, one of pure uric acid reaching the weight of 14 oz.; and this method, which I have employed seventeen times for stone patients, yielded me results which surpassed any before obtained from the lateral operation, considering the size of the calculi removed. In connection with this subject it may be permissible to add here that I have also performed the same operation eleven times for the purpose of removing tumours of the bladder, none of which cases of course appear here, with only one death following the proceeding, viz. from

septicæmia. With such an experience I should never again adopt, in ordinary circumstances, any other form of lithotomy for a large stone in the bladder.

The whole of the calculi extracted by all the methods above named are, with only two or three exceptions, systematically arranged in a cabinet, and have been presented to the Royal College of Surgeons for preservation in the museum there, accompanied by the catalogue referred to.

The following tables summarise the leading facts relating to the sex, age, nature of calculus, operations employed, their results, &c., in regard of the patients who have come under my care both in hospital and in private practice from the first case down to the end of 1889. The last table, giving a general view of the whole, is suspended in the room.

TABLE I.—*Cases operated on in University College Hospital, London, between* 1854 *and* 1874.

Adult males.	Youths and boys.	Girls.	Total number of patients.
79 ...	13 ...	1 ...	93

	Cases of operation.		Deaths.
The 79 male adults were treated—			
24 by perinæal lithotomy, median, medio-bilateral, bilateral, but chiefly by the lateral operation .	24	...	10
55 by lithotrity, by several sittings. A few of these were operated on again at a later period, furnishing in all	63	...	6
The 13 youths and boys were treated—			
10 by lithotomy	10	...	1
3 by lithotrity	3	...	0
The 1 girl[1] was treated—			
1 by lithotrity	1	...	0
Total	101	...	17
Extraction of foreign bodies:			
1 girl lithotomy, supra-pubic.			

[1] This was a patient in the infirmary of Marylebone, the only case of stone occurring there while under my care as visiting surgeon, and hence, ranking as a hospital patient, is placed with the University College Hospital cases.

TABLE II.—*Cases operated on in private practice only between* 1857 *and December* 31*st*, 1889.

Adult males.	Youths and boys.	Females.	Total number of patients.
739 ...	3 ...	14 ...	756

	Cases of operation.		Deaths.
The 739 male adults were treated—			
91 by perinæal lithotomy, median, and medio-bilateral, but chiefly by lateral . .	91	...	33
1 supra-pubic (old) operation . . .	1	...	1
17 supra-pubic (modern) operation . . .	17	...	4
630 by lithotrity, nearly half being at a single sitting. Several cases were operated on a second time, a few a third time, and in six cases four times .	737	...	40
The 3 youths and boys were treated—			
2 by perinæal lithotomy	2	...	0
1 by supra-pubic lithotomy	1	...	0
The 14 women were treated—			
9 by incision, 1 chiefly by dilatation . .	9	...	1
5 by lithotrity and extraction . . .	5	...	0
Total	863	...	79

On examining the above table and the total shown in No. III, which follows, it will be seen that the proportion of children to adults is very small. Among these only 16 males fell to my lot, 13 being in hospital practice; 3 were cases in private—a fresh proof of the rarity of calculus in the children of parents among the middle and upper ranks of life. Sir William Fergusson stated that he had but once received a fee for operating on a child. Deschamps in the latter part of last century stated that he had never seen an example among families in easy circumstances.

The number of females operated on was 15 (14 adults and 1 girl), and of these little need be said here. Hence I shall now deal with male adult cases only, and shall beg you to bear this in mind throughout all subsequent remarks. Deducting these two series of 16 and 15 respectively from 964, the number of male adults remaining is 933, 800 by lithotrity and 133 by lithotomy.

TABLE III.—*Total of all cases of stone in the bladder operated on in hospital and in private practice between* 1857 *and December* 31*st*, 1889.

Total number of cases of operation 964
Total number of patients 849

Male adults—818. Youths and boys—16. Females—15.

	Hospital.			Private.			Total.		
	Number of patients.	Number of operations.	Deaths.	Number of patients.	Number of operations.	Deaths.	Number of patients.	Cases of operation.	Number of deaths.
Male patients (adults):									
Lithotrity	55	63	6	630	737	40	685	800	46
Lithotomy (perinæal) .	24	24	10	91	91	33	115	115	43
Lithotomy (supra-pubic):									
Old method . . .	—	—	—	1	1	1	1	1	1
New method . . .	—	—	—	17	17	4	17	17	4
	79	87	16	739	846	78	818	933	94
Youths and boys:									
Lithotrity	3	3	—	—	—	—	3	3	—
Lithotomy (perinæal) .	10	10	1	2	2	—	12	12	1
Lithotomy (supra-pubic)	—	—	—	1	1	—	1	1	—
Female patients:									
Lithotrity (adult) . .	—	—	—	2	2	—	2	2	—
„ (girl) . .	1	1	—	—	—	—	1	1	—
Lithotomy and dilatation (adult)	—	—	—	12	12	1	12	12	1
							849	964	96
Foreign bodies in the bladder:									
Removed by lithotrite (males)	—	—	—	3	3	—	3	3	—
Removed by supra-pubic operation, old method (female)	1	1	1	—	—	—	1	1	1

Respecting these operations it will be seen that they were performed on 818 individuals, due to the fact that several of the patients operated upon by lithotrity formed fresh calculi subsequently, and required fresh operations for their removal. As before observed, such proceedings have been strictly limited to the removal of considerable formations, evidently newly produced, and not to the recurring concretions already referred to.

Next it should be stated that there were six patients among the entire number who were operated on by me at different periods of their lives and for different stones, both by perinæal lithotomy and by lithotrity, but in each case at a more or less considerable interval of time. These cases are numbered in the catalogue as follows: 170, 233, 341, 396, 474, 714.

Among the 800 cases of lithotrity in the male adult the sum-total of hospital and private practice—

There were 6 patients who had the operation performed four times for different calculi, with considerable intervals of time (several years) between each.

There were 10 patients operated on three times, and 77 patients operated on twice.

Hence there were 592 *patients operated upon by lithotrity once only, at all events by myself;* a very few of these have to my knowledge been operated on a second time by some other surgeon, but almost the entire number have remained free from stone-formation subsequently.

3. NATURE OF THE CALCULI REMOVED.—The calculi removed from male adults in hospital practice were 87 in number, 24 by perinæal lithotomy and 63 by lithotrity = 87.

The calculi removed from male adult patients in private practice are 846 in number, as follows: 91 by perinæal lithotomy, 18 by supra-pubic lithotomy, and 737 by lithotrity = 846.

		Hospital.		Private.		Total.
Uric acid	Multiple calculi .	8 = 53	...	103 = 482	...	535
	Single calculi .	45		379		
Uric acid and phosphate . .		8	...	82	...	90
Oxalate		3	...	29	...	32

	Hospital.		Private.		Total.
Oxalate and urate	4	...	29	...	33
Oxalate and phosphate . . .	—	...	15	...	15
Phosphates	18	...	207	...	225
Cystine	1	...	2	...	3
	87	...	846	...	933

The calculi in 15 female cases (one a girl) were—		
Uric acid	11	
Uric and phosphate	2	
Phosphates	2	
The calculi in 16 cases of male children were—		
Uric acid	10	
Urate and phosphate	3	
Oxalate	1	
Oxalate and urate	1	
Phosphate	1	
	—	31
Total number of cases		964

4. The Age of Male Patients with calculus will be next examined. I have already referred to the very large proportion of elderly men who are affected with calculus, in calling attention to the circumstance that this important fact has been in former time greatly overlooked. The following table forcibly illustrates this view.

Of the entire record of 964 cases, the number of male patients (adults and children) operated on was 949. Their ages are shown in the following table, which presents them in five classes for reasons which will appear.

Class 1 contains all from the earliest age to puberty, say from the first to the fifteenth year.

Class 2, the period from 15 to 25 years, at which stone is most rare.

Class 3, from 25 to 50, during which it gradually becomes more frequent.

Class 4, from 50 to 70, when stone, especially uric acid, abounds.

Class 5 contains all cases above 70 years, when stone is also frequent, but the proportion of vesical phosphatic formations is greater than in the preceding class.

	Below 16 years.	16 to 24 years.	25 to 50 years.	51 to 70 years.	Above 70 years.	Total.
In the hospital	13	5	22	56	4	100
In private	3	8	89	565	184	849
Total	16	13	111	621	188	949

The mean age of the entire adult male cases occurring in hospital and private practice is within a fraction of 62½ years. The greatest age at which I have operated is 91 years, by lithotrity, for a stone of considerable size (Case 797), occurring in January, 1888; the patient, who passes all his urine by catheter, was greatly relieved, and was living (1889) free from his calculous symptoms, and in fair health for his age.

5. NUMBER AND NATURE OF THE FATAL CASES.—I have made it a rule to accept as a "fatal case" any instance in which death took place within six weeks of the operation from any cause; four instances excepted, in three of which it suddenly and instantaneously occurred from failure of the heart's action, the result of long-standing organic disease; the patient in each case being completely convalescent and in apparently good health. In the fourth the death occurred in similar circumstances from acute bronchitis acquired within that period. I am satisfied that this rule is too stringent, but I have preferred to err if at all in accepting a full proportion of deaths.

In considering the question of death it is of course absolutely necessary to deal with children and adults in separate classes. The different degree of risk incurred from lithotomy in childhood and in manhood is so great as to render practically useless any numerical inferences regarding the mortality of cases in which this distinction of age is not kept clearly in view. The number of children is so small in this collection that my remarks will be brief. There were 16 male children and one female. Four were treated by lithotrity, and 13, being mostly large stones, by lithotomy, one of them being very large by the supra-pubic method. I commenced on the principle of employing lithotrity for children whenever

the calculus could be crushed at one sitting, and the very first case of stone which fell to my lot occurred in a girl, and was thus crushed, in 1854.[1] Three other cases followed, the first being in the year 1860, at University College Hospital, all successful. This treatment I enforced at some length in my first work on Calculus, published in 1863, alluding to the practice adopted in the Children's Hospital in Paris, where large calculi were crushed at numerous sittings, with very unsatisfactory results.[2] Among the 13 lithotomies in children there was one death, a case of deformed pelvis from rickets, exhibited at the Royal Medical and Chirurgical Society in 1863, in which with great difficulty I removed the calculus through a preternaturally contracted outlet.[3] Had I been aware of the fact beforehand, I should certainly have performed a suprapubic operation.

Hence I have first to deal briefly with the mortality following 933 cases of operation in the MALE ADULT ONLY, 800 treated by lithotrity, and 133 by lithotomy.

At the middle of the present century, soon after which my series commenced (the first case just referred to dating 1854, although there were practically none, that is only three, before 1860), the relation between lithotomy and lithotrity was that of rival systems for the relief of the calculous patient, the respective claims of which for preference were under consideration by the profession. Sir B. Brodie had declared in favour of lithotrity for cases in which the calculus was small, and the passages favorable and healthy (Royal Medical and Chirurgical Society, 1855). The practice, however, then and for ten years after was to employ lithotomy as a rule, and lithotrity only in exceptional instances. It was much later than this before even half the cases were generally submitted in this country to the crushing operation. When Sir W. Fergusson in 1865 gave a summary of his entire experi-

[1] Vide 'Lancet,' 1854, October 21st.

[2] 'Practical Lithotrity and Lithotomy,' Churchill, 1863, pp. 207—211.

[3] 'Trans.,' vol. xlvii, p. 11.

ence, the total number of his cases was 219, namely, 110 of lithotomy, and 109 of lithotrity—an equal division between the two methods, although the latter had occurred in an increasing ratio during the later years of his practice. My observation of Civiale's practice in Paris, who performed lithotrity in fully seven eighths of the calculous cases which at the rate of about fifty a year passed through his hands, convinced me that this proportion offered far better results than those attained by the English practice, provided Civiale's instruments and procedure, both at that time much superior to our own, were adopted. This conclusion was also shared by Mr. William Coulson, of St. Mary's Hospital, who acted on it towards the end of his career.

But the present relations between lithotomy and lithotrity have gradually been changed. There is no longer any rivalry between the two systems; one operation is complementary to the other. Lithotrity has in fact superseded lithotomy for all ordinary cases of stone, whatever may be the age of the patient; and a cutting operation of some kind is now only necessary or desirable in certain exceptional conditions, extreme size and hardness of the stone being those which chiefly render it necessary.

I commenced practice under the influence of impressions received from Civiale, reserving only my own opinion that lithotomy might occasionally have been adopted with advantage for some of the calculi crushed in Hôpital Neckar at that time. Accordingly, among my first 200 patients, lithotrity was employed in the proportion of about 4 or 5 cases to 1 of lithotomy. In my next 300 it rose to about 8 to 1. And for the last ten years, during which cases of large calculi have been sent to me in an unusual number, the ratio of lithotrity has slightly diminished, the latter five years having furnished 17 cases of high operation in the adult, which, as already said, I have substituted for the lateral with considerable advantage. In relation to this proportion of large calculi it is necessary to note in passing, that one of the results to an

operator of large experience in calculous disease is the attraction to him of advanced and difficult cases. Hence the proportion of patients demanding lithotomy on such grounds increases during the third period of his career, as compared with his experience in the middle and early periods.

But with the large proportion of cases just referred to treated by lithotrity, 800 in number—and let it be remembered that adult cases alone are now referred to,—it necessarily followed that a group of very unpromising patients was formed by lithotomy, differing widely from the average cases formerly operated on by English surgeons, and constituting the bulk of lithotomy records in this country before lithotrity was practised; much also from the lithotomy cases performed by surgeons who submitted only a half or at most two thirds of their patients to lithotrity.

Then it should be further stated here that I have rarely refused to any applicant the last chance of life which an operation might afford him, having done so in fact but five times throughout my career. These were patients who were obviously unfitted by disease and exhaustion to undergo any surgical proceeding whatever.

The group of exceptionally hazardous cases thus set apart in my series for operation by the knife amounted in number to 133, of which 115 were dealt with by perinæal lithotomy, one by the old supra-pubic, and 17 cases by the modern supra-pubic operation.

The series of lithotrity cases comprises 800 operations. Of these, 475 were performed by the old method of one or more sittings according to the size of the calculus, and 325 by the modern method of one sitting only. The mortality, reckoned on the principle laid down above, was as follows:

In 475 of lithotrity by multiple sittings were 33 deaths, or 7 per cent.

In 325 of lithotrity by a single sitting were 12 deaths, or a little over 3¼ per cent.

In 115 of perinæal lithotomy were 43 deaths, or rather over 1 in 3 cases.

The mortality of the 17 cases of supra-pubic operation by the new method was 4 cases; 3 of these occurred in patients whose condition was exceptionally bad: one had been for six years the subject of vesico-intestinal fistula, and his death was certainly not due to the operation, although it was hastened thereby; while the other two would certainly have been rejected by me for perinæal lithotomy, but I gave them the chance of the less formidable supra-pubic operation. But it may be mentioned here that 11 cases of the same operation for vesical tumour already referred to were followed by death in one case only, making 28 cases of the modern supra-pubic operation, as employed for all purposes, in the adult with 5 deaths.

I beg permission here to recall the fact that in reporting the first 500 cases presented to the Society in 1878 I carefully investigated the causes of death, which occurred in 61 cases, and recorded them under several heads in a tabular form. The technical distinctions there employed have been somewhat changed in dealing with the mortality in the 434 cases which have passed through my hands since that date, in accordance with the progress of pathological knowledge. The following table will show the later results in three columns: deaths after lithotrity, after perinæal lithotomy, and after supra-pubic lithotomy.

Causes of death occurring in 434 *male adult cases operated on since preceding report of* 500 *cases in* 1878.

	Lithotrity.	Lithotomy.	Supra-pubic lithotomy.
i. Septicæmia, with deposits in various parts of the body	2	3	1
ii. Acute nephritis, with purulent deposit in the kidneys . . .	3	0	0
iii. Chronic disease of the kidney, with dilatation of the pelvis and ureters	5	1	0
iv. Peritonitis	0	1	1
v. Acute cystitis	0	1	0
vi. Exhaustion in feeble and aged patients, no other cause of death being obvious	3	4	2
vii. Hæmorrhage	0	1	0

	Lithotrity.	Lithotomy.	Supra-pubic lithotomy.
viii. Delirium tremens	0 ...	1 ...	0
ix. Erysipelas	0 ...	1 ...	0
x. In confirmed diabetic patients	1 ...	1 ...	0
	14 ...	14 ...	4

It will be manifest that the death-rate is considerably less in the 433 cases operated on since 1878 than in the 500 cases which occurred before that date, and this in spite of the influx of more formidable cases. This satisfactory result is in great part, although not entirely, due to the increased safety of lithotrity by one sitting, as compared with that by several sitttngs, and to the marked superiority of lithotomy by the supra-pubic route for large calculi to that by the perinæum.

Lithotrity in the male adult.

Series I.—Cases reported to the Royal Medical and Chirurgical Society in 1878 :[1] 422 cases with 32 deaths, mortality 7½ per cent.

Series II.—Cases since that date now reported : 378 cases (325 by one sitting) with 14 deaths, mortality rather over 3½ per cent.

I may note also that among this small number of deaths following lithotrity, Series II, I include one which I was sent for into the country to finish for an aged and worn-out patient (No. 619), whose stone had been already crushed three times by my friend who summoned me, the case being one of unusual difficulty. I emptied the bladder at this sitting, removing a large quantity of calculous matter, and the patient gradually sank from exhaustion. I accepted this case as a fatal one for my own list, certainly not with satisfaction, excepting that which arises from the consciousness of adhering strictly to a principle laid down, however hardly it may sometimes apply.

It is worthy of observation also that no accident in

[1] Vide 'Trans.,' vol. lxi.

operating has been met with in the present series; such as perforation of the bladder by the staff in lithotomy—the breaking of a lithotrite—and the impossibility of withdrawing an over-impacted lithotrite, of each of which an example was described in the first series. The first named was of course fatal; the second and third were successfully dealt with.

6. UNUSUAL CASES.—I shall now very briefly advert to some examples in the collection of calculus formed under conditions rarely occurring, and therefore of unusual interest.

Thus in Case No. 66, an adult, the nucleus of the calculus, which was removed by lithotrity, is constituted by a portion of dead bone, most probably exfoliated from the pelvis. Some years before the operation the patient had been the subject of chronic hip-joint disease, then cured, and considerable exfoliations had taken place from the surface of the hip, several cicatrices being visible.

But I also found a large exfoliation in the bladder, the result of hip disease, in a youth, Case 878, whom I cut by the lateral method, consisting of a great part of the head of the femur thickly covered with phosphates, believing it to be simply a large phosphatic calculus, until subsequent examination showed that it was the bone in question. This must have gradually made its way through the pelvic bone and entered the bladder, remaining there long enough to have acquired a large deposit before symptoms rendered an operation necessary. This case, like the preceding, made a good recovery; both were treated in University College Hospital.

Several examples of encysted calculus have been met with, cases in which the condition was demonstrable at the time of the operation by digital exploration. Two occurred in female patients, and were felt by the medical men present: in one case, No. 883, the stone itself shows by its form the portion which was encysted. This I turned out of its bed by means of my finger without difficulty; the other, No. 887, was almost entirely en-

capsuled, and gave some trouble to remove. Dr. Smith, of Dumfries, who brought the patient to town, was present; both of the patients recovered. Another example in a male subject (No. 193) lay just within the neck of the bladder, and was only felt by me after I had, by lateral lithotomy, removed one from the cavity; when on searching I thought I felt another, but found on introducing my finger into the rectum that it was absolutely fixed, almost the entire formation lying outside the cavity.

In Case 653 two pyriform calculi, each the size of a large nut, occupying a cavity close to the neck of the bladder, were turned out by the finger and a director, Dr. Macnab of Bury St. Edmunds being present.

One very remarkable case is that of a patient æt. 64, No. 714. He was the subject of lithotrity in 1885, having for some years previously passed all urine by catheter. Seven months after, being again a sufferer, and no stone felt by sounding, I explored the bladder by incision from the perinæum, and detected in the neck a number of small calculi in a sac. I opened this by the knife and removed six, each about the size of a large pea, facetted by close contact. I drained the bladder, and the wound healed slowly. Next year his symptoms again became more severe, and the catheter was required every hour. The prostate was very large; phosphatic matter could be felt, but no defined stone. I performed supra-pubic lithotomy, cleared out a quantity of phosphatic matter firmly adhering to the inner coat of the bladder attached by fibrinous deposit, and then established a constant opening, so as to dispense with the use of the catheter in future. Fitted with a well-curved tube and silver plate, he lived for three years in comfort, travelling abroad and taking considerable exercise; never used the catheter again. He died in August, 1889, efficiently served by his tube to the last.

Of course the well-known cases of facetted calculi lying closely packed in front of the bladder, occasionally met with, and composed chiefly of phosphate of lime, are not here referred to, as these are more common than calculi really

encysted within the vesical cavity, which are extremely rare.

I shall refer to the largest calculus in the series, No. 717, as more remarkable for its structure even than for its size. It is composed of uric acid with a small proportion of alkaline base, but without any phosphatic deposit whatever, either internally or externally, notwithstanding that it weighs no less than fourteen ounces. I have never seen in any collection a calculus nearly approaching in size to this *unmixed with phosphates*.[1]

Let me observe that when a calculus has been cut in its largest plane it is easy to trace somewhat roughly the patient's history, reading it, so to speak, from and "between the lines" exposed by the section. Thus a pure uric or oxalic acid nucleus is mostly seen, and if the patient's circumstances permitted him to avoid much exercise, because found by experience to be painful, he escapes cystitis and alkaline urine, and the acid deposit continues. But an attack with muco-purulent urine sooner or later leads to phosphatic deposit, a ring of which appears in the calculus to mark the fact; and another fact, viz. that he was then kept quiet for a time, is indicated by subsidence of the phosphate followed by a fresh ring of uric acid deposit. Similar changes of deposit again appear, and furnish the outline of a history which I have often found interesting, but which cannot be further illustrated here.

I subsequently learned from this patient that as soon as symptoms became painful he assumed the horizontal position, and maintained it night and day for a period of somewhat more than ten years, and thus escaped an attack of cystitis and phosphatic urine. Moreover, the stone is seen to have occupied during, at all events, the latter portion of that period an unchanged position in the bladder, for each side of its base is deeply indented by the flow of

[1] Cheselden's largest calculus weighed seventeen ounces. It is largely composed of uric acid, three separate calculi originally, united to form one through being cemented together by a considerable proportion of phosphatic matter. It is now in the Royal College of Surgeons, No. A.c. 7.

urine issuing from the corresponding ureter, several layers of the crust being thus worn through; illustrating, by the way, the truth of an observation made some time ago, that fresh, pure, healthy urine exercises some solvent power on certain calculous formations. The patient's age was sixty-two when I performed the high operation for him in 1865. He made a good recovery and married about a year after, and wrote me last summer that he was enjoying good health and an active life. He was sent to me by Mr. Atkinson, Bennington, Boston, Lincolnshire.

Among somewhat rare calculous cases should be named three cases of cystine. Of these one was crushed (Case 127) for a gentleman of eighty years of age, who lived to be ninety. Another (Case 690) is the largest I have ever seen; it weighs two ounces and three quarters, and was removed by the high operation in 1884. The patient is living and well. The third (Case 274) was cut by the lateral method in the hospital and made a good recovery.

The two following cases illustrate an incident which seldom occurs. In No. 253 I performed lithotomy for a large uric acid calculus in which spontaneous fracture had recently occurred, producing formidable cystitis. Case 333 was an instance in which fracture of calculus took place on sounding, and a similar case is alluded to in the notes thereon in the catalogue.

Case 7 was that of a man æt. 22, whom I cut in the hospital in 1861; a phosphatic stone which had a piece of sealing-wax about an inch long as its nucleus, which he stated that he had used as a bougie about six months before. He left the hospital, and I saw no more of him until two years ago he called upon me to report himself well, twenty-eight years after the event.

Lastly, I shall only name four cases operated upon for the removal of a foreign body strictly so regarded, no calculous matter being present, since the object had been introduced into the bladder within a few days before coming under observation. Three of these were successfully dealt with by the lithotrite. The fourth, occurring in a

girl in University College Hospital in 1865, I was compelled to deal with by the old supra-pubic operation, the course and results of which appeared to be quite satisfactory, but death suddenly occurred through the bursting of a peritoneal abscess, when she had resumed active habits after supposed complete recovery.

Two were hair-pins, purposely introduced, and two were broken catheters.

Here I must close this somewhat lengthy record. I would gladly have entered on the perhaps more interesting subject of practical lessons in relation to treatment deducible therefrom; but this it was manifestly impossible to do as part of the present paper, and if ever accomplished must form a communication by itself.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 79.)

ON THE

HISTORY OF URIC ACID IN THE URINE,

WITH REFERENCE TO THE FORMATION OF URIC ACID CONCRETIONS AND DEPOSITS.

BY

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The occurrence of uric acid in human urine seems to be somewhat of an anomaly. As a vehicle for the elimination of nitrogen it is not needed. Its place is taken by urea, which adapts itself perfectly, by its bland character and easy solubility, to the mammalian plan of a liquid urine. In birds, serpents, insects, and the great majority of invertebrate species, which void a solid or semi-solid urine, uric acid forms the sole and appropriate medium for the excretion of nitrogen, and is therefore physiologically indispensable. But uric acid is not indispensable to the mammal. The pig is said to void none; and the large herbivorous quadrupeds, during the greater of their life, discharge a urine which is free from uric acid. The information we possess on this subject indicates that the occurrence of uric acid throughout the mammalian class is fitful and inconstant; and the inference seems to be

justified that it is not an essential element in mammalian metabolism. These considerations lead up to the conjecture that the continued presence of uric acid in mammalian urine may be a vestigial phenomenon, analogous to the persistence of rudimentary structures. On this view uric acid should, perhaps, be regarded as a reminiscence of some far distant link in the chain of mammalian descent, and as a remnant of an ancestral path of metabolism, now fallen into disuse, and superseded by a better path, more perfectly adjusted to the requirements of the mammalian type.

But although uric acid be thus physiologically of trivial account, it is, from a pathological point of view, by far the most important component of the urine. It owes this prominence not to any inherent deleterious quality, but to its clumsy behaviour in liquid media. All the trouble with uric acid arises apparently from its sparing solubility, and the sparing solubility and unstable constitution of its compounds. Thereupon depends its tendency to form deposits and concretions, which act as irritating foreign bodies in the tissues and urinary passages. Were it not for the occurrence of these deposits—of sodium biurate in gout, of free uric acid in gravel, and of the amorphous urates as a sediment in the urine—we should not probably be more cognizant, clinically and pathologically, of uric acid than we are of kreatinine, which is voided in about the same proportion with the urine.

The history of uric acid is here considered under the following headings :

1. Spontaneous precipitation of uric acid in normal urine.

2. Composition and reactions of the amorphous urate deposit, and of its natural and artificial analogues.

3. Chemical explanation of the spontaneous precipitation of uric acid in urine.

4. The ingredients in the urine which inhibit or retard the precipitation of uric acid in the normal state.

5. Summary of the history of uric acid within the

urinary channels (*a*) in the normal state, (*b*) in the subjects of uric acid gravel.

6. The factors which determine the occurrence of uric acid concretions and deposits.

1. Spontaneous Precipitation of Uric Acid in Normal Urine.

Uric acid exists in urine in a state of combination with bases as urates. In the course of its transit through the urinary channels it encounters a diversity of physical and chemical conditions, which are calculated to affect the stability of the urates. The urine is generally secreted with an acid reaction; but it is often alkaline, especially after meals. It is sometimes pale, watery, and poor in salts; at other times it is concentrated, rich in salts, and high-coloured. These variations may occur in quick succession, so that there accumulates in the bladder a mixture of urines of all these several characters. In perfect health uric acid maintains itself in solution amid all these various changes, not only so long as the urine is detained in the urinary passages, but even for some time after it has been discharged. But in certain abnormal states this continuity of solution is broken. In the subjects of gravel uric acid is often thrown down in the kidneys or bladder, or is precipitated soon after the urine is voided, while it is cooling, or within an hour or two after emission. Or precipitation may occur somewhat later—in the course of four or six hours, in the form of copious urinary deposits. But this is not all. Numerous observations have led me to the conclusion that every urine which has an acid reaction tends to the eventual liberation of its uric acid. I found that acid urines kept with antiseptic precautions[1] invariably deposited uric acid sooner or later—except when the proportion of that substance was so small that, were it all in the free state, the volume of urine was sufficient to hold it in

[1] This was usually effected by adding a few drops of chloroform to the test-tubes or phials in which the urines were kept.

solution. The time of the occurrence of the precipitation varied greatly. It usually began within twenty-four hours after emission, sometimes in a day or two, and sometimes not for five or six days, or even later. It took place with equal certainty whether the urine was kept in the warm chamber at blood-heat, or was kept at the temperature of the air.[1] The duration of the process varied with the earliness or lateness of its onset. Speaking roughly, urine which began to deposit uric acid in a few hours completed the process in a few hours longer; but if the onset was delayed for some days the deposition of crystals went on slowly for several days subsequently. When the process was at length completed—whether that were early or late—all the uric acid had disappeared from solution. The filtered supernatant urine gave not the least precipitate with hydrochloric acid, nor could there be detected in it, on evaporation to a small bulk and with careful search, any trace of uric acid. This was, at least, the result arrived at with urines of medium density in which free precipitation had taken place.

Neutral and alkaline urines, such as are voided after meals, did not precipitate uric acid, nor any form of urate, however long they were kept. In like manner, urines which were kept without antiseptic precautions, and consequently, after a time, underwent the ammoniacal fermentation, did not precipitate uric acid, unless the occurrence took place early, and before ammoniacal fermentation set in.

We must, therefore, recognise in normal acid urine an inherent tendency to the spontaneous liberation and precipitation of its uric acid. This tendency only assumes a morbid significance when the event occurs prematurely, while the urine is still sojourning in the kidneys or bladder. Viewed in this light pathological gravel may be regarded

[1] In the latter case the urine often threw down amorphous urates. By this occurrence the urates were in some degree withdrawn from the operation of the disintegrating forces; but this only caused delay. Ultimately the deposit changed entirely into crystals of uric acid.

as due to an exaggeration of conditions which prevail, in a less pronounced degree, in the normal state; hence an elucidation of these conditions may be reasonably expected to throw a light on the ætiology of gravel and calculus, and perhaps furnish hints which may be turned to therapeutical uses. Before entering on this inquiry it is, however, necessary to clear up certain points in the chemistry of uric acid and the urates concerning which current views require considerable revision.

2. Composition and Reactions of the Amorphous Urate Deposit and of its Natural and Artificial Analogues.

Uric acid is a bibasic acid; it is represented by the formula $H_2(C_5H_2N_4O_3)$. It forms, like other bibasic acids, two regular orders of salts: namely, neutral urates, with the general formula $M_2(C_5H_2N_4O_3)$; and acid urates or biurates, with the general formula $MH(C_5H_2N_4O_3)$. But, in addition to these, uric acid forms a series of hyperacid combinations of more complex character, of which the hypothetical formula is $(MH[C_5H_2N_4O_3],H_2[C_5H_2N_4O_3])$. To these hyperacid combinations Dr. Bence Jones gave the name of quadrurates.

The neutral urates were obtained by Allan and Bensch[1] by saturating cold solutions of the caustic alkalies, free from carbonate, with uric acid, and boiling down the solution in a retort until crystals made their appearance. The neutral urates are very unstable, and are decomposed in the presence of carbonates, and even by the carbonic acid of the air. The neutral urates are quite unknown except as laboratory products, and their reactions and mode of preparation are such that it is scarcely conceivable that they should ever exist in the animal body, or play any part in the physiological or pathological history of uric acid.

The acid urates or biurates are the most stable and

[1] Liebig's 'Annalen,' Band lxv, p. 184.

best known salts of uric acid. They readily assume the crystalline form, and are easily obtained in a state of chemical purity. They can be prepared artificially under conditions which are germane to those existing in the animal body; and they are encountered pathologically in gouty concretions, of which they form the distinctive constituent.[1]

The quadrurates appear to be much more widely diffused than the biurates. They are present physiologically in the urine, and probably also in the blood. They are unapt to assume the crystalline form, and are difficult to obtain in a state of chemical purity. Their special and distinctive characteristic is that they are decomposed by pure water, with emission of free uric acid. They exist in nature in the form of the amorphous urate sediment of human urine, and as the essential constituent of the urinary excretion of birds and serpents. They can, moreover, be produced artificially under conditions which closely correspond to those prevailing in the living body. These three varieties of quadrurate require separate consideration.

A. *The amorphous urate deposit.*—The amorphous urate deposit has usually been regarded as consisting of biurates—as a mixture of the biurates of potassium, sodium, and ammonium, in varying proportions. This view is, however, quite untenable. The amorphous urate deposit differs essentially in its reactions from the biurates. The biurates are not decomposed by water. They simply dissolve in water, and are again deposited unchanged on evaporation. In order to study the effect of water on the amorphous urate deposit it must first be separated from the other ingredients with which it is mingled in the urine. This is done by filtering off the sediment and washing it on the filter with rectified spirit and drying. If a minute speck of such

[1] Directions for the preparation of sodium biurate in a pure state are given in a footnote to the first section of a paper by the author on the "Chemistry of Gout" which is printed in the present volume.

purified deposit be intimately mixed with a large drop of distilled water and observed under the microscope, it is seen to be slowly decomposed. In a few minutes—five to fifteen—crystals of uric acid begin to make their appearance. These grow and multiply until, in the course of half an hour or an hour, the field of vision is thickly studded with them; and the process goes on, provided the preparation be kept from drying, until the amorphous matter appears to be almost entirely transformed into crystals of uric acid.

Another way of separating the deposit from its associated urinary ingredients is the following. The sediment is taken up in a pipette, and five or six drops are allowed to fall in slow succession, and on the same spot, on a pad of blotting-paper. The liquid parts of the urine are imbibed all round by the blotting-paper, and there remains in the centre a little heap of damp deposit. If a portion of this be picked off on the point of a pen-knife, and examined in the manner described with a drop of water under the microscope, the same scene of transformation will be observed.

This mode of examination—observation of the behaviour of a speck of deposit with a large drop of distilled water under the microscope—will be again adverted to in the course of this paper, and it may be conveniently referred to as the "speck experiment."[1]

It was known to Berzelius and Lehmann that when the amorphous urate sediment was repeatedly washed on a filter with cold water, crystals of uric acid made their appearance on the filter; but it was reserved for Bence

[1] The speck experiment should by preference be performed with distilled water; but ordinary drinking water will generally answer equally well. This is, however, not always the case. Drinking waters are sometimes a little alkaline, and then the demonstration is apt to miscarry. I found that the Manchester pipe water answered the purpose as well as distilled water, but the London water does not do so. The London pipe water—or, at least, that which is supplied to the district wherein I reside by the West Middlesex Water Company—is slightly alkaline from dissolved carbonate of lime, and it acts very slowly and imperfectly on the amorphous urate deposit.

Jones to furnish a rational explanation of the phenomenon.[1] Bence Jones made a minute quantitative analysis of three specimens of amorphous urate deposits, purified by washing with alcohol, and found that the amount of uric acid contained in them greatly exceeded the quantity required to form biurates with the sum of the bases present. He also found, when the purified amorphous urate was treated with water, that a portion of the uric acid was set free and deposited, and that the remainder went into solution as true biurate. Moreover, he succeeded in preparing artificially an imitation of the amorphous urate sediment by dissolving uric acid in weak potash or soda ley, and then adding acetic acid until a slight acid reaction was produced. A dense white precipitate was thus thrown down. This precipitate, when washed with alcohol and dried, presented the properties and reactions of the amorphous urate sediment. It had a finely granular character, it was decomposed by water, and the part which went into solution had the composition of true biurate.

On the ground of these observations Bence Jones concluded that the amorphous urate deposit consisted of a complex compound, in which biurate was united in loose combination with an additional atom of uric acid, and that when the compound was treated with water the loosely combined uric acid was thrown out, and the associated biurate passed into solution. Writing of the potash compound thus artificially produced, he says, "This granular substance may be considered to resemble the quadroxalate of potassa, which differs from the acid oxalate by containing double the amount of oxalic acid, and following this nomenclature it may be called *quadrurate of potassa.*" The formula for this compound according to this view, therefore, would be $KH(C_5H_2N_4O_3),H_2(C_5H_2N_4O_3)$.

Bence Jones, however, qualified his statements respecting the amorphous urate deposit in a way which greatly detracted from their conclusiveness; and this is probably

[1] "On the Composition of the Amorphous Deposit of Urates in Healthy Urine," 'Journ. of Chem. Soc.,' 1862.

the reason why his investigations thereupon have attracted so little attention. He declared that the amorphous urate deposit was not always composed exclusively of the unstable compound which was decomposed by water, but often contained, in addition, a larger or smaller admixture of true biurates; and that in some instances even the deposit was entirely composed of biurates, and did not throw out any uric acid when treated with water. In the final summary of his results he says, "In conclusion, then, it appears that the amorphous deposit of urates in the urine has no constant composition. It is a mixture of different acid urates modified in crystalline form by other substances in the urine. . . . Moreover, uric acid is occasionally found in combination with these acid urates, forming quadrurates, and thus rendering the deposit still more liable to vary in its composition."

In the last three years I have examined a very large number of specimens of the amorphous urate sediment, and have invariably found that they were decomposable by water and exhibited the characteristic reaction with the speck experiment. Sometimes, however, urate deposits are encountered which resist the decomposing effects of water for a considerable time. The cause of this variable resistance to water appears to depend on some kind of contamination. Sometimes it seems due to the varying quantity, and perhaps the varying quality, of the pigment, which adheres so obstinately to the deposits. In other cases it seems to be due to some saline impurity which imparts a slight alkalescence or a slight acidity to the sediment. I doubt whether it is ever due to an admixture of biurates; because, as will hereafter appear, the biurates cannot exist unchanged in normal urine. The only condition in which, so far as I know, true biurate ever appears as a deposit in urine, is when the urine has undergone the ammoniacal fermentation. Under these circumstances biurate of ammonia is thrown down, and may be sometimes recognised under the microscope (mixed with the amorphous and crystalline phosphates) as slender dumb-

bells or globular masses, which are wholly undecomposable by water.[1]

B. *Urinary excretion of serpents and birds.*—Serpents and birds eliminate their nitrogen exclusively as uric acid —in the form of a white semi-solid mortar-like urinary excrement. When this substance is examined in the fresh state under the microscope with a drop of spirit, or of normal urine, it is seen to consist of innumerable minute spheres. These spheres present a radiating crystalline structure, and are, for the most part, about the size of the white blood-corpuscles; some are double this size, and a great many very much smaller. If, instead of a drop of spirit or of urine, the spheres are examined with a drop of distilled water, they are observed to undergo speedy decomposition, with abundant emission of uric acid crystals. If the mode of examination be varied, and the mortar-like substance be treated with a large quantity of distilled water, the same results follow as in the case of the amorphous urate sediment. A portion of the uric acid is set free and remains undissolved, and the rest goes into solution as biurate. The urine of serpents and birds, in fact, is entirely composed of quadrurates, mixed with more or less mucus. The urinary secretion of the large serpents is easily obtained from our Zoological Gardens in large solid masses and in a condition of great purity. There are, however, some necessary precautions to be used in collecting serpents' urine for scientific examination. The excretion should be obtained fresh and uncontaminated with water, and should at once be dried at 100° C. Collected in this way serpents' urine may be preserved in stoppered vessels unchanged for an indefinite period, and

[1] An artificial imitation of the amorphous urate deposit can be produced at will in the following manner. A normal alkaline urine, such as is voided after food, or a urine which is rendered slightly alkaline by the addition of alkaline carbonates or phosphates, is heated with excess of uric acid, and filtered hot. The filtrate is cooled rapidly under a running tap of cold water or on ice. An abundant precipitate falls, which is an exact counterpart of the natural amorphous urate sediment.

furnishes an abundant and almost pure supply of natural quadrurate. The common notion that the urine of serpents and birds consists of biurate of ammonia, mixed with varying proportions of free uric acid, has arisen from the excretion having been collected and kept without precautions, and having, consequently, undergone diverse decomposing changes which have entirely altered its chemical and physical constitution. Qualitative testing indicated that serpents' urine contained no chlorides, phosphates, nor other salts—or only such traces as might be accounted for by the admixed mucus. Small masses of uric acid crystals were occasionally found. The presence of these may be assumed to be due to secondary changes in the quadrurate spheres.

c. *Artificially prepared quadrurates.*—The quadrurates may be produced artificially in a variety of ways. When uric acid is digested at blood-heat with weak solutions of the alkaline carbonates, or of the dipotassic or disodic phosphates, or of the alkaline acetates, it enters into solution as quadrurate. Under favorable circumstances the quadrurate can be directly precipitated from these solutions by rapidly cooling them, especially by cooling them on ice. The quadrurates may also be prepared by treating solutions of the crystalline biurates with the alkaline superphosphates. This latter method involves a reaction which comes into play in the spontaneous precipitation of uric acid in urine, and will be again adverted to. In one or other of these ways I succeeded in producing quadrurates of potassium, sodium, ammonium, calcium, and magnesium.

Potassium quadrurate was prepared by dissolving uric acid in a hot 5 per cent. solution of potassium acetate, filtering hot, and cooling rapidly under a running tap of cold water until a copious precipitate was produced. This was collected on a filter, washed with alcohol, and dried. *Sodium quadrurate* was prepared in a similar manner with the sodium acetate—but the proceedings have to be carried out very quickly, otherwise the product is apt to

be contaminated with free uric acid. *Ammonium quadrurate* was prepared by boiling a gram of uric acid with 200 c.c. of a 1 per cent. dilution of the strong liquor ammoniæ. The solution was filtered hot, and then rapidly cooled. Through the cold liquid an abundant stream of carbonic acid was passed until a bulky precipitate was produced. This was at once filtered off and quickly washed with alcohol. The whole process must be carried out rapidly—otherwise the quadrurate passes into biurate. *Calcium quadrurate* was prepared by dissolving half a gram of uric acid, in the cold, in 100 c.c. of lime water. To the filtered solution acetic acid was added drop by drop until neutralisation was approached. An abundant precipitate was then thrown down, which was treated in the usual way. *Magnesium quadrurate* was prepared by digesting uric acid and calcined magnesia—both in excess—with distilled water at blood-heat, with frequent agitation for about ten minutes. The mixture was filtered warm, and the filtrate immediately cooled under a running tap. A dense flocculent precipitate formed, which was quickly washed with alcohol and dried.

Summary of the properties and reactions of the quadrurates.—The quadrurates present themselves usually as granular amorphous substances. They readily assume the colloidal modification, and when examined under the microscope in this state appear as large translucent globules. The spheres of birds' and serpents' urine are, however, distinctly crystalline, and, as was pointed out by Sir Alfred Garrod, polarize light. Owing to their instability the quadrurates are very difficult to obtain in a state of chemical purity; they are apt, when produced artificially, to be mixed either with free uric acid or with biurates, and in all cases to be contaminated with traces of foreign saline matters. They cannot be dissolved unchanged in any simple menstruum. In is therefore impossible to purify them, as most other substances are purified, by repeated solution and reprecipitation. They are extremely unstable; and they

tend to change in two opposite directions. In weak solutions of the alkaline carbonates or of the dimetallic phosphates they slowly take up an additional atom of base, and are converted into biurates. On the other hand, in water, and in watery solutions of the neutral salts, they are quickly split up into free uric acid and biurate.

The only appropriate solvent of the quadrurates is healthy urine. In acid urines they dissolve pretty freely with the aid of heat, and are again precipitated unchanged on cooling. Such solutions, however, are not quite stable; after a time their uric acid is slowly and at length completely liberated. The quadrurates are much more freely soluble in warm alkaline (not ammoniacal) urines, and in these media they continue permanently unaltered if guarded against septic changes. When such solutions are made at boiling heat, and are saturated, they throw down on cooling bulky deposits which are identical in appearance and reactions with the natural amorphous urate sediment.

With regard to the chemical constitution of these compounds I have adhered to the views and nomenclature of Bence Jones. Quantitative analyses of the artificially prepared potassium and sodium compounds, and of a purified specimen of the natural amorphous urate sediment, yielded to me results which were strictly conformable to the formulæ of the quadrurates above given.

The general conclusions arrived at with regard to the composition and the physiological and pathological relations of uric acid and the urates are concisely exhibited in the following table:

TABLE I.—*Showing the composition and physiological and pathological relations of uric acid and the urates.*

Uric acid . .	$H_2(C_5H_2N_4O_3)$	Not known in the free state in the body nor in the urine, except pathologically as gravel and calculus.
Neutral urates .	$M_2(C_5H_2N_4O_3)$	Not known physiologically nor pathologically. Only known as laboratory products.

Biurates . .	$MH(C_5H_2N_4O_3)$	Known pathologically in gouty concretions. Known in urine only after the secretion has undergone ammoniacal fermentation. Doubtful whether they ever exist physiologically in the body.
Quadrurates	$MH(C_5H_2N_4O_3)$ $H_2(C_5H_2N_4O_3)$	These are specially the physiological salts of uric acid. They exist normally in the urine, and probably also in the blood. They constitute the urinary excretion of birds and serpents. All the morbid phenomena of uric acid arise from secondary changes in the quadrurates.

3. Chemical Explanation of the Spontaneous Precipitation of Uric Acid in Urine.

We are now in a favorable position for considering the chemical reactions which occur in the spontaneous precipitation of uric acid in acid urine. The amorphous urate, or quadrurate, is the only combination of uric acid which can be actually shown to exist in normal urine. The amorphous urate is not unfrequently deposited from the urine on mere cooling. When not thus spontaneously deposited it may be often made to appear by cooling the urine still further by the application of ice. When this is insufficient its presence may be revealed by first evaporating the urine to a small bulk on a water-bath, and then cooling it on ice. By this last method the amorphous urate may be demonstrated to exist even in alkaline urine. Moreover, as I shall presently show, the biurate—the only other combination of uric acid which could conceivably be present in urine—cannot maintain its integrity in normal urine, but is forthwith transformed into quadrurate. The analogy of the urinary secretion of birds and serpents also points strongly in the same direction.

We may therefore, I think, conclude with certainty that the quadrurate is the form, and the only form, in

which uric acid exists in normal urine, and may draw the further conclusion that when uric acid makes its appearance therein in any other guise, such an event is due to secondary changes in the quadrurate.

On the other hand, the urine in which this compound is dissolved is an aqueous fluid, containing, besides urea and pigmentary and other extractives, a number of salts. Among the salts the most important in this connection are the phosphates. These regulate, in the main at least, the reaction of the urine. The phosphates easily oscillate between the monometallic forms (superphosphates) which have an acid reaction, and the dimetallic forms which have an alkaline reaction. When the former preponderate, as is usually the case, the urine is acid; when the latter preponderate the urine is alkaline.[1]

We have, therefore, in an acid urine the quadrurate existing in the presence of water and of superphosphates. These conditions ensure the ultimate complete liberation of the uric acid. The first step in the process is the splitting up of the quadrurate by the action of the water of the urine into free uric acid and biurate. By this reaction half the uric acid is set free. This decomposition is represented by the subjoined equation:

$$\underset{\text{Quadrurate.}}{(MH[C_5H_2N_4O_3],H_2[C_5H_2N_4O_3])} + Aq. = \underset{\text{Biurate.}}{MH(C_5H_2N_4O_3)} + \underset{\text{Free uric acid.}}{H_2(C_5H_2N_4O_3)}$$

But the biurate resulting from this reaction is immediately retransformed, in the presence of superphosphate, by a double decomposition, into quadrurate. Two atoms

[1] It is now generally agreed that the normal acidity of urine is due, not to a free acid, but to the preponderance of acid phosphates. The alkalescence of normal alkaline urines is certainly generally due to preponderance of dimetallic phosphates, and not to carbonates. In the alkaline urines voided after meals I have repeatedly tested the point. These do not usually evolve any carbonic acid when treated with acids. In certain cases carbonates are, however, abundantly present in alkaline urines, especially when carbonates, or salts of the vegetable acids, have been medicinally administered.

of biurate with one atom of monometallic phosphate change into one atom of quadrurate, and one atom of dimetallic phosphate, according to the following equation :

$$\underset{\text{Biurate.}}{2(MH[C_5H_2N_4O_3])} + \underset{\text{Monometallic phosphate.}}{(MH_2PO_4)}$$
$$= \underset{\text{Quadrurate.}}{(MH[C_5H_2N_4O_3],H_2[C_5H_2N_4O_3])} + \underset{\text{Dimetallic phosphate.}}{(M_2HPO_4)}$$

These alternating reactions—breaking up of quadrurate by water into biurate and free uric acid, and recomposition of quadrurate by double decomposition of biurate with monometallic phosphate—go on progressively until all the uric acid is set free.

That these are the actual steps of the process whereby the totality of the uric acid is eventually liberated in acid urines may be deduced from the following considerations and experiments. The first step—the breaking up of the quadrurate into free uric acid and biurate by the action of the water of the urine—is in accord with what has been already shown to be the reaction of water with quadrurates. The occurrence of the second step—the transformation of biurate in the presence of superphosphate into quadrurate—is directly established by the following experiments.

A saturated solution of potassium or sodium biurate is made in hot water and then allowed to cool. When to this solution a strong solution of one of the alkaline superphosphates is added drop by drop, a dense white precipitate is thrown down, which, on examination, is found to possess all the reactions of the quadrurates. A similar result is obtained when the experiment is repeated with an acid urine instead of a solution of superphosphate. If the biurate solution is mixed with about one third of its bulk of an acid urine of medium density, a copious precipitate forms. This precipitate has the usual characters and the reactions of the amorphous urate deposit, or quadrurates. That the result in this latter case is not due to the precipitation of quadrurate pre-existing in the

urine is proved by repeating the experiment with the same urine after it has been deprived of all its uric acid by repeated filtration through uric acid.[1] It still throws down amorphous urate abundantly with the biurate solution.

The transformation of biurate into quadrurate in the presence of superphosphate explains why true biurates never appear as a deposit in normal and undecomposed urine.[2] It also explains why in the spontaneous precipitation of uric acid in urine the process goes on, not merely until a moiety, but until the whole of the uric acid is set free and deposited.

4. On the Ingredients in the Urine which inhibit or retard the breaking up of the Quadrurates in the Normal State.

It has just been shown that uric acid exists in the urine in the form of the amorphous urate or quadrurate, and that when the urine is secreted acid—that is to say, for some sixteen or twenty hours out of the twenty-four—this compound exists amid conditions which, if they stood

[1] It is a curious fact that acid urines are entirely deprived of their uric acid by passing them two, three, or four times through a filter on which a little heap of pure uric acid has been placed. This result is, I believe, brought about in the same way as the spontaneous precipitation of uric acid in acid urines, as already explained; but the process is greatly accelerated by the superadded force of crystalline aggregation. I have given an account of this matter in a paper "On Pfeiffer's Test for Latent Gout," in the 'Lancet' for January 4th, 1890.

[2] This transformation of biurate into quadrurate takes place even in alkaline (not ammoniacal) urines. This was proved by adding a solution of potassium biurate to a urine which had first been deprived of its uric acid by being passed repeatedly through the uric acid filter, and then rendered slightly alkaline by the addition of dimetallic phosphate or bicarbonate. A urine so treated, when evaporated to a small bulk, and then cooled, threw down a dense amorphous precipitate, which possessed the properties of quadrurate, and was decomposable by water.

alone and uncontrolled, would lead to speedy precipitation of uric acid in the free state. But in the normal course no such early precipitation occurs; it only occurs as a remote and postponed event after the urine has been voided. It is obvious, therefore, that the urine contains certain ingredients which inhibit or greatly retard the water of it from breaking up the quadrurates. Were it not for the presence of these inhibitory ingredients uric acid would be thrown out daily in the urinary passages, and everyone would be subject to gravel. Hence an inquiry into the nature of these inhibitory agents has a pathological as well as a physiological interest, and bears directly on the ætiology of calculous disorders. The inquiry is not a simple one. The urine is a very complex fluid. It contains, besides urea, a number of saline constituents, together with pigmentary and other extractives. Where among all these are the inhibitory agents to be found?

Salts of the urine.—Attention was first directed to the saline constituents. It was found that when urine was dialysed, whereby its crystalline ingredients were for the most part removed, it lost to a considerable extent its power of retarding the decomposition of the quadrurates. This observation indicated that the inhibitory power resided, partly at least, in the crystalloids of the urine. The chief crystalloids of the urine are urea, and the chlorides, phosphates, and sulphates of potash, soda, ammonia, lime, and magnesia. Solutions of these several substances in distilled water were prepared, and their effect on the quadrurate was tested by the speck experiment in the following manner. A speck of a purified specimen of the amorphous urate deposit was placed on a glass slide, and intimately mixed with a drop of the solution to be tested. The covering glass was then applied and the result watched under the microscope. The time at which crystals of uric acid began to make their appearance was taken as a measure of the activity of the tested solution in decomposing the quadrurate. The standard of comparison was distilled

water, which usually caused crystals to appear in five minutes. Solutions of urea of various strengths acted precisely with the same speed as distilled water. The chlorides and sulphates, in the proportion of one per cent. and upwards, imparted to water a considerable power of retarding the appearance of crystals. The potash salts were found to have more effect in this respect than the corresponding salts of soda and ammonia. The common disodic phosphate (rendered perfectly neutral to test-paper by the addition of phosphoric acid) showed about the same inhibitory power as sodium chloride. None of these solutions nor any admixture of them approached the natural urine in power of postponing the decomposition of the amorphous urate. More pronounced effects were obtained with the dipotassic phosphate. A solution of this salt containing only 0·2 per cent., and perfectly neutralised, appeared to act almost as slowly on the deposit as a normal acid urine. Urines which were alkaline from fixed alkali had absolutely no decomposing effect on the amorphous urate.

Pigments of the urine.—Attention was next turned to the colouring matters of the urine. The amorphous urates have an intense affinity for urinary pigment; the pigment cannot be removed from them by any solvent which does not, at the same time, destroy their integrity. I had noticed that deeply tinted urates were more slowly decomposed by water than pale-coloured urates. It had also been noticed that artificially prepared quadrurates and the quadrurates which constitute the urinary secretion of birds and serpents, all of which are devoid of colouring matters, are much more quickly broken up by water than the natural amorphous urate, which is always more or less tinted. Moreover, it was found that a urine which had been filtered through animal charcoal, and thus deprived of its pigment, acted very much more rapidly on the amorphous urate deposit than the same urine before it had been filtered through charcoal.

It can therefore scarcely be doubted that the pigments

of the urine play an important part among the ingredients which impart to normal urine its remarkable power of retarding the decomposition of the amorphous urate. In the febrile state, and in other wasting disorders, the urine is sharply acid and rich in urates, and yet such urines are not prone to deposit uric acid, though very prone to deposit amorphous urates. In these cases the urine is always deeply coloured; and the pigments are probably the chief agents which prevent the precipitation of free uric acid under these circumstances.

These observations do not, I think, exhaust this part of the inquiry. It is not improbable that, besides the salts and pigments, there are other components of the urine which contribute to retard the liberation of its uric acid. Moreover, urinary pigments are of several kinds, and they may not be all alike in regard to their power of protecting the integrity of the quadrurates.[1]

5. Summary of the History of Uric Acid within the Urinary Channels; (a) in the Normal State, (b) in the Subjects of Uric Acid Gravel.

Uric acid exists primarily in the urine as a quadrurate. The history of this substance from its birth in the kidneys to its final expulsion—taking a complete cycle from micturition to micturition, with the interposition of a meal—proceeds, as may be gathered from the foregoing observations, on something like the following lines.

(*a*) *In the normal state.*—Starting with a period of fasting, the urine is secreted and accumulates in the bladder with an acid reaction. During this period incipi-

[1] It was observed in the case of artificially prepared quadrurates and of serpents' urine—both of which are either free or comparatively free from organic admixtures of any kind—that weak solutions (containing 0·1 per cent. to 0·2 per cent.) of the alkaline bicarbonates or dimetallic phosphates slowly decomposed them and threw out uric acid, in spite of these solutions having a distinctly alkaline reaction with litmus-paper. Such a result never followed in the case of the natural amorphous urate deposit.

ent decomposition of the quadrurate goes on with slow liberation of uric acid, but the process does not go far enough to induce actual precipitation. Then follows a meal and digestion of food. This is attended with a change in the reaction of the urine, which now becomes alkaline. As the alkaline stream descends into the bladder, the contents of that viscus become first neutral, and at length alkaline. During this period the decomposition of the quadrurate is arrested, and the previously liberated uric acid is recompounded into quadrurate. As the effects of the meal pass off, the acidity of the urine is restored, and the collected product in the bladder, when finally voided, presents a neutral or slightly acid character; and the quadrurate contained in it is discharged in its original state of complete, or almost complete, integrity.

(*b*) *In the subjects of uric acid gravel.*—Starting as before with a fasting state and an acid urine, the process of uric acid liberation proceeds more rapidly, and results in actual precipitation of crystals in the kidneys or bladder. In the slighter cases of the disorder, and in the milder phases of the more severe ones, the deposited crystals are redissolved on the advent of the alkaline tide after a meal, or they are swept out of the bladder at the next micturition. No permanent concretions remain, and no calculous symptoms are engendered, or only slight and transient renal pains. In severer cases the deposited crystals fail to be entirely dissolved by the alkaline tide; on the contrary, they aggregate into minute but permanent concretions in the kidneys. This event marks an adverse change in the risk of precipitation. The already formed concretions operate—according to a well-known law of chemical physics—as soliciting foci, and give a great additional impulse to the tendency to precipitation. The concretions thus go on increasing until at length the phenomena of renal gravel are fully developed. In severe cases of gravel the alkaline tide after meals is sometimes markedly reduced in strength, or even, as I have occasionally observed, entirely abrogated.

6. The Factors which determine the Formation of Uric Acid Concretions and Deposits.

The more remote and predisposing causes of urinary precipitations do not come within the scope of the present inquiry.[1] Whatever these causes may be, they must be translated into changes in the composition of the urine before they can determine the occurrence of calculous accidents. No amount of morbid proclivity to uric acid gravel can take effect if the urine be alkaline, nor if the proportion of uric acid in it fall below a certain point. The causes which will be here considered are those which lie exclusively in the chemical constitution of the urine itself.

In the preceding section proof was given that the salines and pigments of the urine exercise a protective influence against premature precipitation of uric acid; and it may hence be inferred that a diminution of these salines and pigments may sometimes act, in a negative manner, as a determining factor in the production of gravel and stone.

Poverty in the salines of the urine is probably an influential factor in the disproportionate frequency of stone among the children of the poor as compared with the children of the easier classes. The prevalence of stone among the natives of India is also probably to be explained in the same way. The children of the poor are fed largely on farinaceous articles, bread, gruels, oatmeal, and potatoes,

[1] It would be of some interest to ascertain whether anatomical deviations in the kidneys, such as exaggerated pouching of the infundibula or calyces, do not sometimes act as predisposing causes of renal gravel. If such abnormalities occur, they would obviously occasion undue detention and stagnation of the urine in the purlieus of the kidney, and thus give opportunity to a tendency, otherwise insufficient, to determine deposition of uric acid. Moreover, slight anatomical differences of this kind between the two kidneys might account for what is so often observed, namely, the unilateral incidence of the symptoms of renal gravel. So far as I know this point has not hitherto been investigated.

with but a scanty allowance of milk, meat, and fish. Wheat-flour contains only 0·51 per cent of mineral matter in proportion to the totality of the dry substance; oat-meal only 2·50 per cent.; potatoes only 2·50 per cent.; whereas milk contains 5·50 per cent., and the various forms of meat and fish 5 to 5·50 per cent. Rice, which forms so large a part of the diet of the natives of India, only contains 0·39 per cent. of mineral matter in proportion to the totality of the dry substance of the grain. These enormous differences in the amount of saline ingredients in the articles of food must, of course, make a corresponding difference in the proportion of the saline constituents of the urine. On the other hand, the well-known immunity enjoyed by sailors from stone and gravel depends, no doubt, as Mr. Plowright has shown, on the prodigious quantity of salt which seafaring men habitually consume with their food. The same observer has pointed out that the dwellers in a district of Norfolk called Marshland, where the drinking water is brackish, are singularly free from stone, as compared with their less fortunate neighbours in the adjacent districts of that county.[1]

Deficiency of pigment in the urine.—In chronic Bright's disease with contracting kidneys the urine is conspicuously pale, and is often indeed almost entirely devoid of pigment. There is no excess, but rather a diminution of uric acid in the urine in these cases; nevertheless deposits of uric acid are by no means uncommon, and sometimes actual renal gravel occurs. The percentage of salines is also low, and this doubtless contributes to the result; but probably the prepotent factor in the pre-

[1] See a paper by Mr. C. Plowright, of King's Lynn, in the 'Medical Times' for October 10th, 1885. Mr. Plowright, on the evidence of some experiments by Mr. H. C. Brown, attributes the good effect of salt to its alleged property of increasing the solvent power of water on uric acid. This is, however, I am satisfied, on the ground of very exact determinations both by myself and others, not the correct explanation. The real action of the salt is, I believe, as a retarder of the decomposition of the quadrurates.

cipitation of uric acid in these cases is the deficiency of pigment in the urine.

Poverty of the urine in salines and pigments, however, only accounts for certain limited groups of calculous cases. There are other and larger groups in which the urine is neither defective in salts nor in colouring matters. The subjects of calculous disorders among the easy classes—especially those of a gouty type—usually void a urine which is full-coloured and abundantly rich in salts. In these cases the chief determining factors—and the only two which I shall here consider—are the grade of acidity of the urine, and the proportion of uric acid contained in it. The speck experiment was found unsuitable for investigating the influence of these factors. For this purpose another mode of experimentation—one that approximates more nearly to the conditions of the actual clinical problem—was adopted. In the beginning of this paper I drew attention to the fact that all urines with an acid reaction precipitated their uric acid sooner or later, and the inference was drawn that this inherent tendency was the same in kind (though less pronounced in degree) as the tendency existing in actual gravel. On this view it might be reasonably conjectured that whatever helped or hindered in the one case would equally help or hinder in the other case—in other words, that the conditions which hasten or retard the precipitation of uric acid in a sample of urine preserved in a test-tube would, if they could be made applicable, hasten or retard the precipitation of uric acid in the urinary passages. The proceeding followed was to charge a series of test-tubes each with 10 c.c. of a normal acid urine. One of these was a control tube, and had no addition made to it. To the others additions were made of known quantities of various substances, of which it was desired to know the effects on the time of onset of uric acid precipitation. The contents of the tubes were protected from decomposing changes by the inclusion of a few drops of chloroform. They were then corked and kept in the warm chamber at blood-heat. The tubes

were frequently examined, and the time when uric acid began to be deposited was noted. When the experiment was finished the acceleration or postponement of precipitation in the several tubes, as compared with the control tube, was computed.

Grade of acidity of the urine.—The degree of acidity of the urine exercises, as might have been expected, a potent influence on the time of precipitation of uric acid. In some cases of gravel I found the acidity of the urine fully twice as high as the normal average. In two such cases the urine as tested by the speck experiment was found to act on the purified amorphous urate as rapidly as distilled water; but when the acidity was reduced to its normal level by the addition of sodium carbonate, it had then no more power in this respect than healthy urine, showing clearly that in these cases the determining factor in the disorder was solely excess of acidity. It was also found, experimenting in the way described above, that the addition of an exceedingly minute quantity of an alkaline carbonate postponed the time of precipitation very notably. The following table displays some of the results obtained by this method:

TABLE II.—*Showing postponement of precipitation of uric acid by the addition of minute quantities of alkaline carbonates to the urine.*

Additions made to the urine.	Time when uric acid began to be precipitated.	Postponement of precipitation.
No. 1. Urine alone—control tube .	2 hours	—
„ 2. Urine + 0·04% Pot. Bicarb. .	4 „	2 hours
„ 3. Urine + 0·04% Sod. Bicarb. .	5 „	3 „
„ 4. Urine + 0·04% Lith. Carb. .	10 „	8 „

The quantities of the alkaline carbonates added in this

experiment were so small that the reaction of the urine, as tested by litmus-paper, was not sensibly affected; and yet the postponement of precipitation was very considerable—considerable enough, had the events occurred in the urinary passages, to make the difference between the occurrence and non-occurrence of gravel. It will be observed that the sodium carbonate acted more powerfully as a retarder than the potassium carbonate, and that the lithium salt acted more powerfully than either. This, however, was solely due to the difference in their atomic weights. When these salts were used in quantities proportionate to their saturating power no difference could be detected between them. It need scarcely be said that if the carbonates were added in sufficient quantity to render the urine neutral or alkaline no precipitation of uric acid took place.

When the neutral salts, chlorides, sulphates, and phosphates were tested by this method, the results obtained were conformable to those obtained by the speck experiment. The potash salts were found to be superior as retarders of uric acid precipitation to the soda salts, but all were incomparably inferior in this respect to the carbonates.

Proportion of uric acid in the urine.—It is a common notion that uric acid gravel depends simply on an excess of uric acid in the urine; and the frequent appearance of copious sediments of this substance in the urine in such cases naturally lends support to this presumption. I am, however, not aware of any reliable analyses which show that the subjects of uric acid gravel have always or even generally a higher percentage of uric acid in their urine than other persons, or that they render a larger amount per day. It is at any rate certain that individuals may habitually discharge a urine rich beyond the average in uric acid, and yet be quite free from the symptoms of gravel.

Cases are sometimes encountered in which the urine is caught, as it were, in the very act of depositing uric acid

gravel—cases in which the urine, as it is voided, sparkles with crystals of uric acid. On four occasions I have been able to estimate the uric acid in such urines. The results are shown in the following table:

TABLE III.—*Showing the percentage of uric acid in four urines which were in the act of depositing uric acid at the time of emission.*

No. 1	contained	0·084	per cent.	of uric acid.
„ 2	„	0·076	„	„
„ 3	„	0·032	„	„
„ 4	„	0·022	„	„

In the first and second cases the percentage of uric acid greatly exceeded the average; in the third case it was slightly below the average; and in the fourth case it was greatly below the average.

The average proportion of uric acid in normal urine is about 0·04 per cent., and during the prevalence of the alkaline tide after meals it often runs up to 0·10 per cent. —without, of course, involving any risk of precipitation. It would, therefore, appear probable that in clinical gravel the concurrence of other favouring conditions — high acidity and poverty in salines and pigments—is of more importance than mere excess of uric acid.

The general results of this part of the inquiry may be summed up in the following propositions. The conditions of the urine which tend to accelerate the precipitation of uric acid are—(1) high acidity; (2) poverty in salines; (3) low pigmentation; and (4) high percentage of uric acid. And conversely, the conditions which tend to postpone precipitation are—(1) depressed acidity; (2) richness in salines, especially of potash salts; (3) richness in pigments; and (4) a low percentage of uric acid. On the interaction of these factors the occurrence or non-occurrence of gravel appears to depend; and probably the most important of these factors is the grade of acidity.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 87.

A STUDY OF FIFTY CONSECUTIVE CASES

OF

OPERATION FOR THE RADICAL CURE OF NON-STRANGULATED HERNIÆ.

BY

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A STUDY of fifty consecutive cases of operation for the radical cure of non-strangulated herniæ, including all the first fifty I have performed, cannot, I venture to think, fail to be of some interest, although the ultimate results of the procedure should not be pronounced upon finally until several more years have elapsed. I have undertaken this study, I believe, without any strong preconceived notion, and simply with a view to learn as much as possible from the series for my own future guidance, and to impart the facts learned as fully as may be to others interested in the subject.

The surgical interest of this procedure centres, I think, round the following considerations:

1. Is it called for?

2. Is it safe—(*a*) as regards the patient's life; (*b*) as regards the contents of the scrotum?

3. Does it secure against a return of the hernia?

These are all vital questions, which can only be answered when we have had a much larger experience. In the meantime nothing can contribute so much to our forming an accurate estimate of the place which this class of operation is to take among the recognised procedures of surgery as the careful analysis and publication of completed series.

In putting these cases together I have purposely excluded operations for radical cure performed on herniæ already strangulated. The latter belong to a totally different category. And although my statistics would be, in some respects, favorably influenced if these were included, it has been considered more advisable to keep them out.

To the first question, whether operations for the radical cure of herniæ are called for, I think most surgeons nowadays would feel inclined, for a large group of selected cases, to give an affirmative answer. The dangers of ruptures, especially among the less favoured members of the community, are amply shown by the large number which are daily operated on for strangulation. The discomforts of the condition, too, and the disabilities entailed upon those suffering from it, are also so well known as to need no comment.

We must all admit, then, that some means of getting rid of herniæ are urgently called for in some cases at all events, if the cure can be accomplished with safety to the patient. Indeed, it may be urged that a certain amount of risk may be accepted by the patient afflicted with this defect and who desires an operation for his cure, seeing that, if nothing is done, he has daily to face the dangers of strangulation.

Admitting, then, that the cure of this condition is most desirable, what are the risks as to life which the patient has to face who is anxious for an operation for the radical cure of a non-strangulated hernia?

I think upon this point the series of cases now before us gives clear evidence as far as it goes.

There has not been a single death among the whole

fifty operations, though some were very troublesome. And though in my next series of fifty additional cases, which will not be complete for some time, there will be at least one death, occurring in a peculiarly complicated case, the fact that in half a hundred operations on patients suffering from all the various forms of hernia not one death has occurred, shows that under the newer conditions of wound treatment the risks of operating for the conditions in question are very small.

Let us now glance at the question whether the state of these patients immediately after the operation and during the healing of the wound was at any time critical.

And first as regards suppuration. It is well to be clear as to what is meant by one of these wounds suppurating. In the first place, if a stitch is left a little longer than necessary and a drop of discharge forms round it independently of the rest of the wound, which has healed by first intention, this ought not to be described as a case of suppuration of the wound. Such a state of things after a hare-lip operation would not be so called.

Again, if a drain-tube is used and is left under a dry dressing say for ten days or a fortnight, and some soft aseptic lymph is then found in it or in its track, the rest of the wound being soundly healed, this, I presume, should not be called a suppurating case.

But if, on the other hand, any part of the wound has failed to unite, and from out of the field of operation pus is escaping even in small quantity, such a case is here described as suppurating, whether the temperature shows a corresponding rise or not.

Of such cases as the last there are in this series only two, Nos. 39 and 41: both of these suppurated beyond question, although they did well otherwise, and were none the worse of the delay in healing. All the rest healed by first intention. In No. 15, after union had taken place two deep sutures were discharged from a small chink at the upper end of the incision, and their escape was preceded by a few drops of discharge. In No. 30 the wound had

healed *per primam*, and the stitches had been taken out on the seventh day; but the patient was a very wild little fellow, who was constantly pulling the dressings off and romping about, until he was at last tied down in the bed. Before this, however, on the eleventh day he had to some extent forced open the edges of the recently healed incision, and of course this spot had to granulate up, which it did rapidly.

There were, then, only two *bonâ fide* cases of suppuration. This is undoubtedly two too many. But the fact that thirty-nine cases were operated on before the first wound broke down, and that after the forty-first all healed as one would have desired, justifies the hope that in a second series of cases still better results may be obtained.

Having got rid of suppuration, almost all other sources of anxiety were eliminated after operation. Shock was not noticed; hæmorrhage was of course absent; and nothing was seen of the accidental wound infections, such as erysipelas, pyæmia, &c. As to the temperature in these cases, it varied; but in many cases it rose a few degrees within the first thirty-six hours, especially in the case of children, even where everything was otherwise satisfactory. In No. 6, an exceedingly fat and intemperate woman, an attack of jaundice with rheumatic or gouty swellings of some of the joints retarded convalescence, but the state of the wound at the umbilicus gave no anxiety. In No. 15 pneumonia appeared on the day after operation, and ran a normal course in nine days.

These and one of measles are the only cases of intercurrent affections to be noted.

Accidents during operation occurred in two cases. In both after the sac had been tied, but before the rings were closed, a sudden struggle of the patient forced down several coils of intestine between the patient's thighs. The gut was in each case washed, dried, and reduced without difficulty, and not the least ill effect followed.

The duration of time until the removal of the skin

stitches was usually from the tenth to the fourteenth day, at which time healing may be said to have been complete and firm in nearly all cases. A few cases required a longer, but most a shorter period.

From this survey it would appear that the risks to the patient's life and general health from operations for the radical cure of hernia may be very slight indeed if due care is observed.

As regards the contents of the scrotum, no ill effects at all, either to the cord or testis, were observed to follow this operation so far. But it is interesting to note that in two cases with double herniæ the patients had been operated on by Mr. Wood on one side by the subcutaneous method before they came to me for operation on the opposite side. In both complete atrophy of the testicle was found on the side operated on by Mr. Wood, the other testicle remaining normal up to date.

The ages of the patients ranged from three months to seventy years. Three were under six months; two between six months and one year; thirteen were between one and five years; six between five and ten; one between ten and twenty; eight between twenty and thirty; two between thirty and forty; six between forty and fifty, and one over seventy.

Several varieties of rupture are included in this series. The largest number were inguinal, as might be expected. Of these, thirty were on the right, ten on the left side, including double herniæ, of which there were four cases.

Of umbilical herniæ there were three, of femoral two cases—one right, the other left. The cæcum was found in the sacs of two right inguinal cases.

The list includes forty-two patients, of whom four had double herniæ, and four were operated on a second time after recurrence. In these last four cases of re-operation the rings were sutured, in two with silk, in one with chromic gut, and in one with kangaroo tendon at the first operation.

The operation performed for inguinal herniæ was in twenty-eight cases the same. It was one designed by the

writer several years ago, and has been sufficiently described elsewhere. Five cases of recurrence are credited to this operation as now performed.

In one case Macewen's operation was done.

In the three umbilical cases I adopted in two a measure also designed by myself and published some years ago. One remained firm two years after operation, the second recurred.[1] In the third a less elaborate procedure was followed, and in this case recurrence was speedy.

Of the femoral cases, my own method of securing the stump of the sac was employed in one; in the other the latter was simply reduced within the ring, which was closed with silk sutures.

In most of the earlier cases the carbolic spray was employed throughout the whole proceeding, but not for the last year or two, and nothing has been lost by the omission, all other precautions to secure asepsis having been taken with increased care.

Drainage, too, has become less and less necessary as the details of drying the wound before the last act of suture has become better understood. I rarely now go beyond leaving a strand of twisted silk in the lower angle of the wound until the first dressing is removed, and then only when there has been an extensive dissection. As a rule the stitches in the skin are all inserted before the sponge is removed from the wound, and if the latter is then seen to be quite dry no sort of drain is used, and the threads are knotted firmly. In the majority of cases the first dressing on its removal about the tenth day has been found practically quite dry, and I always regard myself now as having been very remiss in some detail if such is not the case.

For the deep sutures in the rings I venture to think that carbolised silk ought always to be preferred. It is strong, easily sterilised, pleasant to work with, and gives a very secure knot. If it is tied too tightly round the

[1] Patient heard of since writing; is now said to be quite well (August, 1890).

fibrous tissues which it includes, it occasionally, however, works to the surface ultimately, even if perfectly sterile; but this is not common. On this point the present series of cases gives some interesting evidence. But it must be remembered that all these patients were up and about a few weeks after operation without any truss, and many undertook very heavy labour soon after leaving bed, and also without truss.

In Nos. 2, 13, 15, 46, 48, and 50, one or more deep stitches worked their way to the surface, but without giving any further trouble at a period ranging from the thirteenth day to the ninth month. Considering that at least 200 deep stitches must have been left in the tissues in these fifty cases, the percentage which came away may be considered small. I think the fault has usually been attributable to using too much force in tying them on the included tissues, and for this reason I am now content if the edges of the openings are simply brought firmly together. If this is so the percentage of stitches which come away in the next series will probably be smaller.

As regards the efficiency of these operative procedures as a means of preventing the return of ruptures, we must wait for a final judgment until a much longer time has elapsed. But as far as they go I have spared no pains to find out the ultimate result. This, as is well known, is a difficult matter with hospital patients, who often change their abode, and cease to present themselves for examination in spite of urgent requests to do so. While writing this paper I have posted cards to the last known addresses of all the patients in the list whose condition has not been recently examined or heard of, and have embodied the results in the appended tables.

From these it appears that the results of thirteen operations cannot be ascertained at all, as the eleven patients on whom they were done cannot be traced since leaving hospital.

In eight the hernia returned, and in four of these a second operation was done with complete success so far as

is known. In one of the cases of recurrence kangaroo tendon was the material used in the first operation, for the rings, but it dissolved so rapidly that the rupture came down almost before the patient left hospital. In another, chromic gut was employed for the first operation. In the remaining two, silk was the material for suture. In the four other cases of recurrence no second operation has yet been undertaken. Two of them were umbilical herniæ, two inguinal.

In the remaining thirty there has been no recurrence when last seen or heard from: five were well and without hernia at between forty and fifty months after operation; seven between thirty and forty months; six between twenty and thirty months; six between ten and twenty months; and six between two and ten months.

In many of these cases the sutured part had, as I have already said, been subjected to severe strain, almost immediately after leaving bed. "Plate-laying" and "quarrying" have been undertaken in this way without bringing back the hernia, and "whooping-cough" and bronchitis have been passed through soon after operation without ill effect on the sutured area.

Except in the cases of umbilical herniæ all these patients have been told *not* to wear trusses on leaving bed, and only in two cases has this rule been departed from, for special reasons. In one or two of the other cases I should have recommended a truss had I known that the ruptures were returning under severe strain.

In not one of all my own cases have I ever seen or heard of any ill effects upon the contents of the scrotum.

In every case here set down the operation was only done when all the other usual means of controlling herniæ had been tried and failed for one reason or another; and this is likely to remain my own guiding rule for the present. There may be exceptions to it, but I think we must be careful about admitting them. If I had operated in all cases in which I have been requested to do so my list would have been much longer.

If I have learned anything from this study I think it may be summed up as follows:

1. That the operation, if great care and attention to detail is observed, may be performed with very little risk of any kind.

2. That the spray ought to be dispensed with as very chilling, and not giving more security against sepsis than can be provided in less troublesome ways.

3. That drainage ought as a rule to be rendered unnecessary by careful handling of the tissues during dissection, so as not to bruise them and leave shreds likely to necrose, and also by arrest of all oozing before final closure of the wound, which should be dried out at the very last moment.

4. That trusses are not needed in the great majority of cases after operation, but should be ordered for those who have evidently very weak abdominal walls, and who are obliged to return to very heavy work, if there be the slightest sign of recurrence of the hernia.

No.	Name.	Age.	Nature.	Date of operation.	Operation.
1	R. Foster	44	Right oblique inguinal for 10 years	April 2nd, 1884; discharged April 24th	Sac opened; omentum removed; pillars sutured, strong catgut; drain-tube; spray; gauze dressing
2	Emily Huntingford	42	Umbilical, 5 years; pain, sickness	April 1st, 1885; discharged May 6th	Abraded skin included in two curved incisions and removed; ring size of forefinger; omentum and sac cut away; ring doubly sutured with eight silk sutures; drain-tube; iodoform and salicylic wool dressing
3	James Swanson	1½	Right oblique inguinal, acquired when 2 months old	June 17th, 1885; discharged July 1st	A. E. B.'s operation; silk sutures; gut drain; salicylic wool dressing
4	Anne Marshall	70	Left femoral for 40 years	July 8th, 1885; discharged Aug. 3rd	Sac opened; omentum cut away and gut reduced; neck of sac tied with catgut; femoral ring closed with two strong catgut sutures; skin stitched with gut; gut drain; Lister's gauze dressing
5	Charles H.	3½ mos.	Right oblique inguinal, acquired 3 weeks after birth	July 11th, 1885; discharged July 18th	Sac isolated with some difficulty; divided between two chromic gut sutures, but lower part not removed; one stitch was put into the pillars to draw them together (? material of suture); spray all the time; salicylic wool dressing; gut drain
6	Maria Tuck	46	Umbilical irreducible for 10 years	Sept. 2nd, 1885; discharged Oct. 21st	Same operation as No. 2; catgut drain; spray; skin sewed with chromic gut

Healing process.	Remarks.	Result.
Wound healed without accident. Temp. only touched 100° on 10th day	—	Could not be traced after leaving U. C. H.; house at address given pulled down.
1st dressing 3rd day, bloody serum only. 2nd dressing 6th day, only serum, feels very well. 3rd dressing, drain removed, also skin stitches. 4th dressing 15th day, brown serum. 5th dressing 16th day, no discharge. 6th dressing 19th day, one deep stitch came away at lower angle of wound. Left U. C. H. well on May 6th. Temp. only exceeded 100° on 3rd day, and fell on escape of serum from 101·2°; did not touch 100° afterwards. Convalescence free from anxiety	Not such a fat patient as is common in cases of umbilical hernia	Examined March 24th, 1887; no trace of hernia. Wears abdominal belt.
1st dressing 3rd day, no discharge, drain removed; quite well in every way. Temp. only touched 100·8° on 3rd day; after this was usually below 100°	—	Examined March 19th, 1887; no trace of hernia; no atrophy of testicle; child quite well.
1st dressing 7th day, wound healing by first intention. 2nd dressing 11th day, wound quite healed. Convalescence uninterrupted. Temp. did not once touch 100°	—	Could not be traced, though written to.
1st dressing 3rd day under spray; all well except soreness of skin of buttocks from carbolic acid; temp. 102·6°; next day temp. 98·4°, child quite well. 2nd dressing 5th day, wound healed up to gut drain, which was removed. 3rd dressing 7th day, wound perfectly healed. 8th day left hospital quite well	—	The father of patient, who is a medical man, says there is no trace of return of hernia, *i. e.* in Dec., 1889.
Attack of jaundice and gouty swellings commenced 4th day, with temp. 101·4°. Edge of dressing raised on 8th day; some serum escaped from drain opening; not further disturbed. Hypostatic pneumonia on 13th day, lasting a few days. Wound dressed daily, quite free from all inflammatory reaction. By Oct. 7th wound healed, except drain opening. Patient well	A very fat patient of intemperate habits; was very difficult to manage during convalescence. Although wound did not close at once there was no suppuration, but a drain of serum, evidently from clots of blood contracting and organising in wound	For some months no return of hernia. I think I heard casually from a friend of patient that it has since come back. Patient has since had gouty attacks like that described. (Note since writing. — Patient heard of lately; is quite well.)

No.	Name.	Age.	Nature.	Date of operation.	Operation.
7	Fred. Ashworth	9	Left inguinal, oblique, acquired 6 years	Oct. 13th, 1885; discharged Nov. 4th	Sac not down; pillars sutured with silk, skin with chromic gut; salicylic wool dressing; spray
8	F. Ashworth. Re-operation	9	Same hernia	Jan. 8th, 1886; discharged Feb. 1st	Sac divided between two silk ligatures; stump reduced within ring, which was then sutured with silk. During dissection one or two of the former ligatures were found; no trace of irritation about them. Catgut drain
9	John White	1½	Right inguinal, oblique, acquired at 6 wks. of age	Oct. 21st, 1885; discharged Nov. 13th	Sac divided between two silk ligatures; pillars sutured with three silk ligatures; catgut drain
10	Walter White	10 mos.	Right oblique inguinal, congenital	Nov. 13th, 1885; discharged Dec. 1st	—
11	John Foale (double)	4	Right inguinal, acquired 3 weeks after birth	May 7th, 1885; discharged May 22nd	Sac opened, then ligatured in two places, and divided between; stump reduced; pillars sutured with two catgut stitches; drain-tube and silk sutures in skin; spray; gauze dressing
12	John Foale	4	Left inguinal, acquired 3 weeks after birth	Dec. 11th, 1885; discharged Dec. 25th	Sac divided between two silk ligatures; pillars closed with three silk sutures; skin sewn with catgut; catgut drain; spray; salicylic wool
13	Walter Hunt	1½	Right inguinal, congenital, noticed when 6 weeks old	Jan. 8th, 1886; discharged Jan. 20th	Sac divided between two silk ligatures; stump reduced; rings closed with silk sutures; catgut drain
14	Richard Greenfield	5 mos.	Left oblique inguinal, congenital	Feb. 5th, 1886; discharged Feb. 24th	A. E. B.'s operation; sac divided between two silk sutures, the upper of which was then used to draw the stump within the inner ring and close the latter; then three other silk sutures in walls of inguinal canal; spray; salicylic wool

Healing process.	Remarks.	Result.
1st dressing 8th day, seemed to have healed by first intention. 2nd dressing 11th day, slight moisture, boracic lint. 3rd dressing 16th day, no moisture. Temp. never touched 100° from beginning to end	—	Hernia returned almost immediately owing to bad cough.
1st salicylic wool dressing removed on 12th day, wound healing by first intention. 2nd dressing 14th day, still healing. 3rd dressing 19th day, drain removed. 4th and last dressing 23rd day, wound quite healed. Temp. rose to 101° on operation day, and next night to 100·6°; the next to 100°, remaining below this until discharged	—	No return of hernia on March 26th, 1886, when patient was last seen. Could not be traced after this; letters in Jan., 1890, returned.
1st dressing on 2nd day. 3rd on 6th. Temp. about 100° until 7th day, when it rose to 101°; cause undiscovered; again on 15th. Child remained well throughout	—	Patient could not be traced; letters returned 1888.
—	—	Patient could not be traced; letters returned 1888.
1st dressing 5th day, drain removed. 2nd dressing on 7th day, stitches in skin removed, edges of wound "a little sloughy," (?) testicle swollen. 3rd dressing 12th day; the wound has healed up except where drain-tube lay. 4th dressing, wound healed except at lower angle. Not dressed again	On 2nd day temp. reached 103°, on 3rd 102·6°, but afterwards was below 100° until patient was discharged	No return in Nov., 1885, in spite of bad bronchitis. Cannot be traced; letters returned Jan., 1890.
1st dressing 5th day, hardly any moisture, drain removed. 2nd dressing 7th day, wound nearly healed. 3rd dressing 8th day, stitches in skin removed. Temp. rose to 103·4° on evening of operation, and next night to 102·4°. After this went down, and uninterrupted convalescence ensued	—	No trace of recurrence of hernia on Nov. 16th, 1888.
1st dressing 5th day, wound healing. 2nd dressing 11th day, stitches in skin removed, a small moist spot remains where drain lay; dressed with boric lint, moist. Temp. 101° on night of operation and next night, then normal until discharged	On March 9th a deep stitch came away; spot of moisture soon healed	No trace of return of hernia on Nov. 12th, 1888. Cannot be traced; letter, Jan., 1890, returned.
st dressing, wound healed by first intention. 2nd dressing changed for wool and collodion. Stitches in skin removed on 12th day. Discharged on 18th day quite healed. Temp. rose on 2nd night to 101·2°, on 3rd to 100°, then remained below 100° until discharged, except on 15th day, when it was 100·4° and 101·6°	No truss was worn at any time after operation	No trace of return of hernia when examined on Dec. 24th, 1888, in spite of recent bad whooping-cough. Quite well when examined Feb. 26th, 1890.

No.	Name.	Age.	Nature.	Date of operation.	Operation.
15	William Self	20	Left inguinal, acquired 2 years ago	Feb. 27th, 1886; discharged April 4th	A. E. B.'s operation; three sutures in ring. T. vaginalis also opened; stitched up. Varicocele operated on at same time; excision of portion between ligatures; no spray; Hyd. Bichlor. to wash wound; drain-tube
16	Harry Cooper	5	Right inguinal, oblique, acquired 4 years ago	March 3rd, 1886; discharged March 15th	A. E. B.'s operation; lower part of sac in this case removed, as it was small and had come out of scrotum; five sutures in rings; catgut drain; spray; c. gauze
17	William Pride (double)	1½	Right inguinal, congenital; large with wide ring	March 16th, 1886; discharged	A. E. B.'s operation; four silk sutures in rings. Just after tying sac and dividing it, patient struggled, and the ligature slipped, a couple of feet of bowel protruding between the thighs; reduced easily, no further trouble; catgut drain; spray broke down in middle of operation
18	William Pride	1½	Left inguinal, acquired six months ago	March 26th, 1886	Same operation; four silk stitches in pillars; catgut drain
19	George Pairvell	1½	Right inguinal, acquired	May 28th, 1886; discharged June 14th	A. E. B.'s operation; sac removed; no drain; spray
20	Mary A. Shay	1	Right inguinal, congenital	Dec. 15th, 1886; discharged Dec. 30th	A. E. B.'s operation; sac removed
21	William Whitbread	7	Right inguinal, congenital; large ring	September 2nd, 1886; discharged September 18th, 1886	A. E. B.'s operation; 5 stitches in rings; spray

Healing process.	Remarks.	Result.
1st dressing on 2nd day, coverings having slipped; tube shortened. 2nd dressing 5th day. 3rd dressing 6th day, the last having slipped; tube removed for good. 4th dressing 8th day, stitches in skin removed, healing by first intention; some swelling and redness remains. 10th day, a little pus can be squeezed out of chink of upper angle. 13th day, two ligatures came away from pillars. April 4th, perfectly healed; discharged	The 2nd day patient developed pneumonia of right base, with temp. ranging from 103° to 98·4°. Attack over in 9 days, and normal temp. from this on. At no time was patient's condition at all critical. He might have left U. C. H. sooner, but had to await turn for convalescent home	June 7th, 1886.—No return of hernia. Has been at work. There is still a tiny track leading to a ligature which has still to come away. Cannot be traced; letter returned Jan., 1890.
1st dressing 7th day, wound dry, drain removed. 2nd dressing 13th day, wound healed *per primam* everywhere	Temp. only rose to 100·2° on 2nd day, and on 4th to 100°, afterwards normal	March 18th, 1887.—No return of hernia on coughing or straining; health excellent. Cannot be traced; letter returned Jan., 1890.
1st dressing 5th day, wound healthy. 2nd dressing, wound healed; stitches in skin removed, and most of catgut drain. April 2nd, circumcised	Temp. first night 101·4°. 2nd, 100·6°, afterwards from 98·6° to 100·6°. Child's convalescence uninterrupted. The protrusion of the bowels did not seem to produce any ill effect	Nov. 16th, 1888.—No return of hernia. Died of diphtheria at beginning of 1889 at home.
st dressing 12th day; 2nd, 13th, on account of wetting with urine which has produced eczema over abdomen. Discharged 17th day, wound being perfectly healed	Temp. 101° evening of operation; normal on and after 4th day. No untoward symptoms after operation	Nov. 16th, 1888.—No return of hernia.
st dressing 5th day, wound almost dry. Stitches in skin removed. Wool and collodion. 2nd dressing 8th day, wool and collodion. 3rd dressing 11th day, wool and collodion. 4th dressing 14th day, healed by first intention	Convalescence uninterrupted. Temp. on 4th day 101·4°, which was the highest recorded	March 19th, 1887.—No return of hernia; no atrophy of testicle. Cannot be traced; letters returned Jan., 1890.
st dressing 6th day, healed *per primam*. Wool and collodion	Temp. 101° on 2nd day	March 18th, 1887.—No return of hernia; health excellent. Cannot be traced; no address left.
st dressing 14th day, healing. 2nd dressing 17th day, healed *per primam*. Discharged to-day	Convalescence uninterrupted. Temp. 101°, highest recorded	August, 1888.—No return of hernia. Child quite well.

No.	Name.	Age.	Nature.	Date of operation.	Operation.
22	Fred. Hammond	2	Left inguinal, congenital	August 26th, 1885; discharged September 2nd, 1886	Sac opened; ligatured with silk in two places, divided between; Stump reduced; rings closed with 2 chromic gut stitches; no drain
23	Fred. Hammond. Re-operation	2	Left inguinal; recurrence	August 20th, 1886; discharged September 2nd, 1886	A. E. B.'s operation; silk used everywhere this time; one suture for sac, two for rings; catgut drain
24	Chas. Andrews (double)	4½	Right inguinal, congenital	October 22nd, 1886	Sac tied at neck and divided; stump reduced; rings closed with 3 silk sutures; no drain; spray
25	Arthur Westley	3 mos.	Right inguinal, congenital in 1st week	September 15th, 1886; discharged October 6th	Sac divided between two fine silk sutures; stump reduced; rings drawn together with *one* kangaroo tendon suture; spray
26	Arthur Westley. Re-operation.	7 mos.	Left inguinal, congenital in 1st week; rings wide	January 4th, 1887; discharged January 29th, 1887	A. E. B.'s operation; three silk sutures; spray; no drain
27	Arthur Westley	10 mos.	Right inguinal; recurred	April 22nd, 1887	A. E. B.'s operation; four silk sutures; no drainage; spray
28	Mary L. Beach	41	Umbilical; acquired 9 months ago	Jan. 5th, 1887	Sac, skin, and omentum cut away; ring closed with five stout silk sutures

Healing process.	Remarks.	Result.
1st dressing 6th day, healed *per primam*. 2nd dressing 8th day, stitches in skin removed. Child sent home	Temp. 100·2°, only once; convalescence uninterrupted	Reappeared April, 1886.
Union *per primam*	Convalescence uninterrupted	October 24th, 1888.—No return of hernia in spite of whooping-cough, which has produced a rupture on opposite side.
1st dressing 5th day, everything quite dry. 2nd dressing 8th day, stitches in skin removed. Healing *per primam*. Wool and collodion	Highest temp. 100·4°	November 12th, 1888.—No return of hernia, but is getting a rupture on the opposite side, which was operated on by Mr. Wood at King's College Hospital. There is also almost complete atrophy of the testicle on this side. No return of hernia when seen Feb. 23rd, 1890; opposite testicle atrophied, other quite normal.
1st dressing 2nd day, free serous discharge. 2nd dressing 4th day, wound doing well. 3rd dressing 5th day, some pus (?) came out of wound. 4th dressing 6th day, no pus. 5th dressing 7th day, no pus. 6th dressing 8th day, wool and collodion. Discharged with wound healed on 19th day	Temp. 2nd day, 102·4°, and remained over 103° all night. On 3rd day temp. 100°	Returned after June, 1887; see below, No. 27.
1st dressing 10th day, wound healthy. 2nd dressing, wound healthy, some orchitis. Wound had to be dressed daily on account of wetting with urine. Stitches in skin removed on 14th day. Discharged well 21st day	Temp. reached 103·4° on 12th day, but convalescence was good.	
1st dressing 2nd day on account of soaking with urine; wound looked quite well; no serous discharge. 2nd dressing 7th day on account of urine; wound dry. 3rd and 4th dressings on 9th and 11th days for the same reason. Two stitches in skin removed on 15th, the remaining ones on 18th. Wound soundly healed	Temp. rose to 103° on 2nd day. Dressings soaked with urine. Convalescence uninterrupted without suppuration in spite of urine. Stitches removed by 18th day; wound soundly healed	No return of either hernia, June 14th, 1887.
1st dressing 7th day, wound healed *per primam* without trace of pus. Stitches removed 14th day, all quite dry. Discharged quite well on 23rd day	Hernia size of large orange, 3 inches by 4. Vomited first two days. Patient very fat. Temp. did not touch 100° during convalescence	Patient became enormously stout. Hernia returned by October, 1887, but not large in October, 1888. Cannot be traced; letters returned, 1890.

No.	Name.	Age.	Nature.	Date.	Operation.
29	Alfred Cooke Beach	6	Right inguinal, acquired when two years old	Jan. 5th, 1887; discharged Jan. 23rd	A. E. B.'s operation; spray
30	Fred. McGregor	5	Left inguinal, acquired; large ring	Feb. 2nd, 1887; discharged March 1st	Pillars stitched at once with three silk sutures, as sac had gone back; no drain
31	Samuel Craker	23	Right oblique inguinal, acquired 9 years ago	April 6th, 1887	Macewen's operation, using silk instead of gut; spray
32	Sidney Hudson	2	Right oblique inguinal, since birth	April 26th, 1887; discharged May 10th	A. E. B.'s operation; four silk sutures; catgut drain; spray
33	Sidney Hudson. Re-operation	2	Right oblique inguinal	Oct. 28th, 1887; discharged	A. E. B.'s operation, as before; spray and drain-tube
34	Alfred Johnson	21	Right inguinal, congenital	April 27th, 1887; discharged May 28th	Operation as usual, except that sac was not divided below the point tied (? correct notes)
35	Frederick Newman	25	Right oblique inguinal, acquired 6 weeks ago	June 23rd, 1887; discharged	A. E. B.'s operation; four silk sutures; spray

Healing process.	Remarks.	Result.
1st dressing 13th day, wound doing well; stitches removed. Wool and collodion. Discharged well 19th day. Wound healed *per primam*	Temp. rose to 101° on 2nd day, then fell to normal	Nov. 12th, 1888.—No trace of return of hernia on right. On the left, operated on by Mr. Wood three years ago, the testicle is quite atrophied. Quite well when seen on Feb. 23rd. No trace of hernia, 1890.
1st dressing 7th day, wound healed *per primam;* stitches in skin removed. 2nd dressing 10th day, wound not so well, as boy is very wild and pulls dressings off. 3rd dressing 11th day, the edges have been burst open by romping. 13th day, wound granulating; boy tied down in bed. 18th day, wound healing. March 1st, discharged well	Except for rise of temp. to 103·4° on 12th day, probably due to iodoform, there is nothing to note about convalescence	Wound soundly healed. No return of hernia when seen on Feb. 27th, 1890.
1st dressing 2nd day; all quiet; 2nd dressing 13th day. Union *per primam* complete. Stitches in skin removed	Convalescence absolutely free from any abnormal symptom. Patient had been operated on for strangulated hernia two years ago	Rupture bulging to some small extent owing to heavy work. Worked without a truss always since operation. Truss ordered. Slight bulging when seen Feb. 23rd, 1890. Truss worn.
Dressings changed on 2nd, 4th, 5th, and 10th day. Union *per primam* everywhere	Convalescence free from any abnormal symptom	Rupture returned in about 4 months (no truss was worn). Cannot be traced; letters, Jan., 1890, returned.
Detailed notes mislaid	—	Cannot be traced since operation. Letter, Jan., 1890, returned.
1st dressing 9th day, wound healed *per primam;* all but two stitches in skin removed. 2nd dressing 13th day, the two remaining stitches had cut a little. 3rd dressing 15th day, wound dry and healed; 21st day out of bed	Temp. did not rise above 100·4° all the time	Returned for examination Feb. 23rd, 1890. Says hernia came down soon after leaving hospital. Has worn no truss since. Very large hernia. Very weak abdominal wall.
1st dressing 15th day, wound healed perfectly *per primam.* Stitches in skin removed	Convalescence perfectly free from any abnormal symptom; 5 mos. later one of the silk ligatures worked its way to the surface; all of the others remained quiescent	April 15th, 1889.—No return of hernia. Wears a truss, as his work as a "platelayer" is heavy. Letter, Jan., 1890, not replied to.

No.	Name.	Age.	Nature.	Date.	Operation.
36	Ellen Barker	6	Right oblique inguinal	July 1st, 1887	No sac found; rings closed in usual manner with three silk sutures; spray; no drain
37	James Stephens	22	Oblique inguinal, acquired	Aug. 10th, 1887; discharged Aug. 29th	A. E. B.'s operation; spray
38	Fred. Keeble (double)	4	Right inguinal, congenital; rings large	July 19th, 1887; discharged August 3rd	A. E. B.'s operation in usual way
39	Fred. Keeble	4	Left inguinal, congenital; ring large	Aug. 27th, 1887; discharged Sept. 27th	A. E. B.'s operation; five silk sutures in rings; lower part of sac left in scrotum; a few coils of gut were coughed out before sac was closed; easily reduced; no drain
40	J. W. H—, naval engineer. Private case	33	Right inguinal, congenital	Sept. 3rd, 1887; discharged Sept. 21st	A. E. B.'s operation; two strong silk stitches in ring; much omentum cut away after ligature in seven or eight places; no spray; lower part of sac closed by stitches to form tunica vaginalis; drain-tube
41	Sarah Parmer	39	Right femoral, acquired 7 years ago	Sept., 1887	A. E. B.'s operation; omentum adherent; removed with sac
42	Harry Gibson	4	Right oblique inguinal, acquired at birth	Nov. 15th, 1887; discharged Dec. 3rd	A. E. B.'s operation; five silk sutures in rings; silk drain
43	William White	2	Left inguinal, acquired when 6 weeks old	Nov. 11th, 1887; discharged Dec. 6th	A. E. B.'s operation; four silk sutures in rings; silk drain
44	W. F. Pearce	45	Right inguinal, acquired 16 years ago	Mar. 29th, 1888; discharged April 20th	A. E. B.'s operation; sac removed; spray; no drain

Healing process.	Remarks.	Result.
1st dressing 7th day; wound healed perfectly *per primam*; stitches in skin removed; same dressing applied	Convalescence perfectly free from any abnormal symptom	Nov. 19th, 1888.—No return of hernia and no other trouble.
Wound healed *per primam*	Temp. 100° same night; normal next day; reached 100° on 3rd, 4th, and 5th day; after this below 100°	Cannot be traced; left no address in hospital. No reply to letter, Jan., 1890.
Wound healed *per primam*	—	Cannot be traced; left no address. No reply to letter, Jan., 1890.
1st dressing same evening; lower angle of wound opened to allow escape of serum. 2nd dressing 2nd day; lower stitches removed to permit free drainage. No old tenderness. 3rd dressing 3rd day. 4th dressing 5th day; wound not looking at all well; discharge of pus free; all stitches in skin removed; fomentations. After this did well	Temp. 103° on evening of operation, and next day 104·6°, then 102° 3rd day. On 24th day wound was granulating well, and all bad symptoms were gone. On 31st day wound quite superficial. Went home	Ditto.
Wound healed *per primam* under 3 dressings	Temp. rose two degrees day after operation; was subsequently normal. Convalescence free from any bad symptom	Nov. 2nd, 1888.—No return of hernia. Is back at duty. Is much pleased with result.
Healed *per primam* under two dressings	No abnormal symptoms of any kind. This had been a very troublesome rupture before operation	Nov. 12th, 1880.—No trace of hernia in spite of chronic cough. No reply to letter, Jan., 1890.
1st dressing 11th day, wound quite healed	Highest temp. 101° on 4th day. Convalescence free from abnormal symptoms	Cannot be traced. Letter, Jan., 1890, not replied to.
Dressings 2nd, 3rd, 5th, 9th day, when suppuration took place. By the 20th day all suppuration had ceased. On 26th day went home quite well	Temp. 101·4°	Father writes, Feb. 23rd, 1890:—"No return of hernia; boy quite well."
1st dressing 9th day; wound looked well. 2nd dressing 16th day; wound healed *per primam*. Stitches in skin removed. Truss applied. Sent home on 23rd day	Highest temp. 100·2°. No abnormal symptoms	Writes, February 23rd, 1890:—"Has no return of hernia in spite of very heavy work. Wears no truss."

No.	Name.	Age.	Nature.	Date.	Operation.
45	W. Beasley	40	Right inguinal, acquired 10 years ago; rings large	Sept. 7th, 1888; discharged Sept. 25th	A. E. B.'s operation; omentum adherent to sac and removed with latter; no spray; silk drain
46	Agatha Minniford	20	Right oblique inguinal, acquired 5 years ago	Sept. 19th, 1888; discharged Oct. 3rd	A. E. B.'s operation; four silk sutures in ring; silk drain
47	George Hardy	9 mos.	Right inguinal, congenital; ring very large	Nov. 2nd, 1888; discharged Nov. 24th	A. E. B.'s operation; six silk ligatures in rings; silk drain; no spray
48	William Fulcher	25	Right oblique inguinal, acquired 7 months ago	Jan. 16th, 1889; discharged Feb. 6th	A. E. B.'s operation; seven silk sutures in rings; no spray; no drain
49	William Coates	10	Right inguinal, acquired	Feb. 26th, 1889; discharged March 8th,	A. E. B.'s operation; body of sac removed in this case; five silk sutures in rings; silk drain
50	John Thomas	21	Right inguinal, acquired 4 years ago	March 1st, 1889; discharged March 26th	A. E. B.'s operation; very large ring; six silk sutures to close them; no spray; no drain

Healing process.	Remarks.	Result.
1st dressing 4th day; drain removed, very little serum. 2nd dressing 8th day; accidental wetting with urine; wound doing well. 3rd dressing 11th day; wound quite healed *per primam*. Stitches in skin removed. Sent home 14th day	Temp. on day after 101·6°; afterwards normal	Cannot be traced; letters returned Jan., 1890.
1st dressing 9th day; wound dry; silk drain removed. 2nd dressing 13th day; wound perfectly healed *per primam*. Went home on 17th day quite well	Highest temp. 100·4°. No trace of any abnormal symptom	Writes, Feb. 25th, 1890: —"No return of hernia. Some deep sutures came away some weeks after leaving U. C. H. Now quite well. Wears truss by another surgeon's advice."
Dressed 2nd, 3rd, 4th days on account of wetting himself. 5th day drain came away quite dry; wound quite healed. 6th day measles developed and ran usual course. Slight impulse on coughing	—	Cannot be traced; letter, Jan., 1890, not replied to.
1st dressing 3rd day, wound looks well, quite dry. 2nd dressing 9th day, wound healed *per primam*; one stitch had cut a little. All stitches in skin removed. Dressings left off on 18th day; quite healed	Highest temp. 100·6°, 26 hours after operation; after this it was normal.	Examined Feb. 23rd, 1890. Hernia has returned under very heavy work without truss. Some stitches are working out during last fortnight.
1st dressing 6th day, wound healed and dry. Silk drain removed. 2nd dressing 8th day, two stitches in skin removed. 3rd dressing 11th day, remaining stitches removed	Temp. rose to 100·6°, 26 hours after operation, then fell for good	Cannot be traced; letter, Jan., 1890, not replied to.
1st dressing 8th day, union *per primam*. 9th day, stitches in skin removed. Healed soundly	Temp. 101·6° 8 hours after operation, then below 100° to end of case. At end of 1889 a stitch worked its way to surface	Seen January, 1890. Hernia is returning; stitches are working out.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 94.)

SALICIN COMPARED WITH SALICYLATE OF SODA

AS TO EFFECT ON THE

EXCRETION OF URIC ACID, AND VALUE IN THE TREATMENT OF ACUTE RHEUMATISM;

WITH SOME DEDUCTIONS AS TO THE CAUSATION OF THE DISEASE.

BY

A. HAIG, M.A., M.D.Oxon.

Received March 10th—Read April 22nd, 1890.

It seems to be a pretty general opinion that salicin is not nearly so useful in acute rheumatism as salicylic acid or salicylate of soda.

Dr. T. J. Maclagan has noticed this general impression ('Lancet,' i, 79, p. 875), and sought to explain it by suggesting that the specimens of salicin used were not pure, and more recently ('Lancet,' i, 1890) he has maintained that the dose of salicin in general use is not large enough.

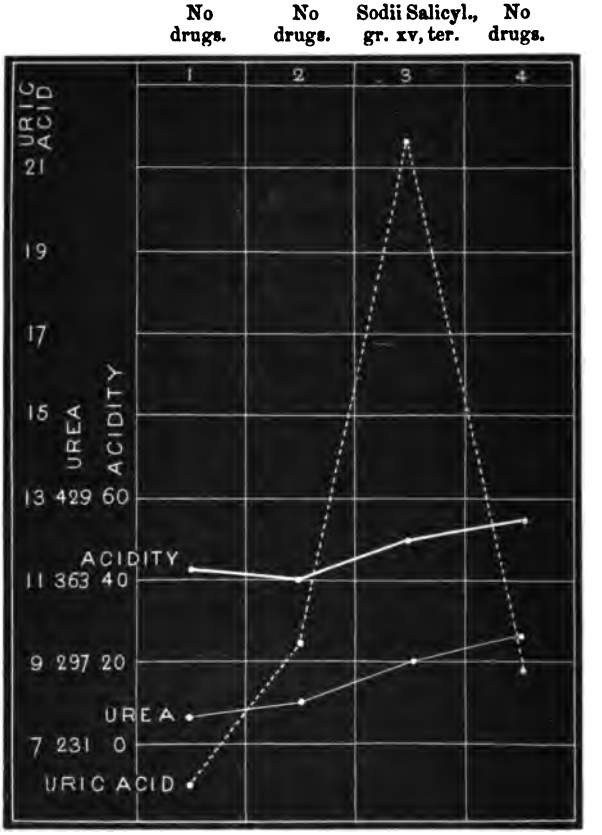
Dr. Brunton ('Pharmacology and Therapeutics,' p. 939) says of salicin, "Its action is less powerful than that of salicylic acid, and its depressing effect on the circulation less marked."

I have myself also noticed apparently great differences in the effects of salicin and salicylic acid both in health

and disease. In consequence of these observations I was led to make comparative experiments to test the effects of these drugs on the excretion of uric acid, and I propose in this paper to bring forward some of my results.

Fig. 1 shows the excretion of uric acid under salicylate of soda taken to the extent of 45 grs. in the twenty-

FIG. 1.



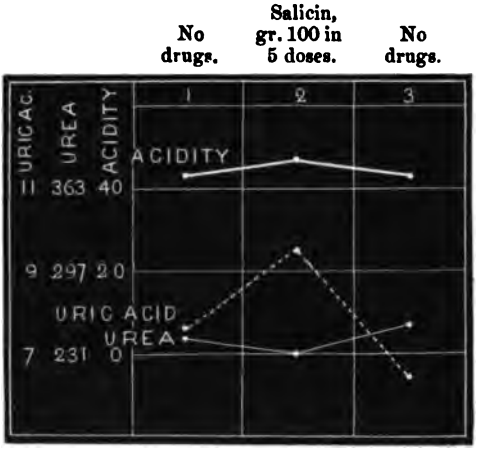
Uric acid excretion by salicylate of soda.

four hours. It is the ordinary curve of excretion obtained with this salt or with salicylic acid, and corre-

sponds very well with the figure on p. 136 of my paper on salicylic acid and its salts in vol. lxxi of the Society's 'Transactions.'

Fig. 2 is on the same scale as Fig. 1, and shows the excretion of uric acid on a day when 100 grs. of salicin were

FIG. 2.



Showing that 45 grs. of the sodium salt has six times the excretive power of 100 grs. of salicin, or weight for weight thirteen times the power.

taken in the twenty-four hours. I have several times before got similar results, showing the greatly inferior excretive power of salicin as compared with a salicylate. And there is this further point of difference as regards the action of the two substances: the urine on day 3 of Fig. 1 gave with perchloride of iron a purple colour so dark that no light would pass through it in a test-tube; while on day 2 of Fig. 2 the reaction was much less marked, and light passed easily through it. It happened fortunately that the amount of urine on these days was the same within 20 c.c., so that there could be no error on the score of dilution; and I have noticed the same thing several times before, viz. that salicin has much less effect on uric acid and gives

perchloride of iron in the urine after salicin as compared with an equal weight of a salicylate.

A most interesting series of cases from the Leeds General Infirmary showing the inferiority of salicin as compared with salicylic acid in acute rheumatism is published in the 'Lancet,' vol. ii, 1876, p. 254.

The late Dr. Hilton Fagge ('Lancet,' ii, 1881, p. 1031) drew up some valuable tables on the comparative treatment of rheumatic fever by mint water, alkalies, and salicyl compounds; these show that with mint water patients got well on the seventh to the eleventh day, with alkalies on the fourth to the ninth day, and with salicylates on the second to the sixth day, and I think the experience of others will now fully bear out his conclusions in favour of the salicylates.

In my paper on salicylic acid in the 'Transactions' of the Society previously referred to (vol. lxxi, p. 137) I have said that the important point in the action of salicylates is that they appear to be able to render the excretion of uric acid independent of acidity, a point in which, so far as I know, they stand alone amongst drugs; and in my thesis for the degree of M.D. ('Brit. Med. Journ,' July 7th, 1888) I have said that their curative action in rheumatism seems to me a strong point in favour of the uric acid causation of this disease. It now seems to me that the fact I have just been pointing out, viz. that salicyl compounds are active in the cure of acute rheumatism exactly in proportion to their power over the excretion of uric acid, is another and by no means a weak argument in the same direction.

I propose now to examine shortly some of the best known and attested facts with regard to the action of drugs and diet in acute rheumatism, and see how far they will bear out the supposition of uric acid causation.

To begin with diet: is there any fact better known and more completely attested than that a lowly nitrogenous or non-nitrogenous diet is of the greatest importance in acute rheumatism? and is there any adequate explana-

tion to be given of its effects, except its influence on the formation and excretion of uric acid?

Among other authors Bouchard ('Leçons sur les maladies par ralentissement de la nutrition,' pp. 241–2) narrates in a most interesting passage that children fed much on meat, meat juice, and jelly suffer from gastro-intestinal derangements, constant affections of the skin, and early migraine; and he goes on to say, "Le rhumatisme avec ses manifestations diverses est précoce et grave."

With regard to migraine I have the best of all reasons (viz. personal experience) for endorsing this opinion, and I believe that meat produces rheumatism by producing and accumulating uric acid just as it produces migraine.

Look again at the effects of beer, of excessive muscular exertion or exposure to cold and damp,[1] and do they not all affect uric acid, and affect it in just the same way both in rheumatism and gout?

As to treatment, look at alkalies; do they not represent next to salicylates by far the most successful treatment of rheumatic fever? And what is their action on uric acid? just like salicylates, they cause a *plus* excretion of uric acid both in health and disease,—only in the latter, having first to overcome the acids present, their action is slower and less powerful than that of salicylates. And in accordance with this we see from Dr. Fagge's table that a large number of patients get well on the second day or sooner with salicylates, but not till the fourth day or later with alkalies.

Some interesting points in this connection have been brought out by those who have used acids or substances which raise the acidity in the treatment of acute rheumatism.

In the 'Lancet,' i, 1874, p. 231, is recorded a case in which Dr. Wilks gave dilute nitro-hydrochloric acid ℥ xv, *quartis horis*, with milk, bread, and beef-tea as a diet. I will only note two points in the history of the case: first, that

[1] "Collective Investigation Report," 'Brit. Med. Journ.,' i, 88, p. 387.

on the ninth day of treatment the patient was observed to be perspiring freely, but the temperature was still as high as 101·2°; second, that five days later (14th day) the swelling of the joints had gone and the urine was alkaline.

I would also refer to some interesting remarks by Dr. Fuller in the 'Lancet,' ii, 1862, p. 669, where he points out that ammonia does not act as an alkali, and does not relieve rheumatic fever, and that under its use the urine remains acid and the pains bad; but when potash is substituted for it within two days the urine becomes alkaline, and the pains are much relieved.

The action of ammonia in this matter, and the fact that it acts as an acid and not as an alkali, is now well known; and anyone who is interested in the point will find the facts well stated in Dr. Mitchell Bruce's book on 'Materia Medica and Therapeutics,' 4th ed., p. 48.

This no doubt explains the value of a dose of Sp. Amm. Aromat. in the uric acid headache, which has been pointed out by myself and others; that is to say, it acts like an acid, and it raises the acidity of the urine very decidedly, as I have plenty of curves to show.

It will not be supposed that nitro-hydrochloric acid made the urine alkaline (though I do not deny that indirectly by causing dyspepsia it might do so); how then did Dr. Wilks' patient get well even on the fourteenth day?

From Dr. Fagge's table we see that most of the mint water patients got well on the seventh to the eleventh day, and if mint water had no action at all nitro-hydrochloric acid must have had a bad or adverse action.

The explanation is, I think, that rheumatic fever is a self-curing disease, and with favorable circumstances, rest in bed and low diet, tends to recovery.

To illustrate this point a little let us suppose that some one, who is estimating his urinary excreta from day to day, goes to bed and puts himself on milk diet; what will be the result on the excretion? His urea will fall, say from 500 grs. to about 300 grs.; his uric acid, which was pre-

viously slightly below its natural amount in proportion to urea, having a relation of 1 to 35 or 1 to 38, will tend to rise above it, having a relation of 1 to 28 or 1 to 26 (*i. e.* a *plus* excretion of uric acid); and the acidity of the urine, previously equal to say 60 grs. of oxalic acid, will fall to about 40—45 grs. If in addition he puts on plenty of blankets so as to keep the skin moist, there will be a further fall of acidity, and a further tendency on the part of the uric acid to rise and be excreted in excess of the urea: we have here, in fact, a natural *plus* excretion of uric acid under the influence of alkali, *i. e.* of the lowered acidity of the urine and increased alkalinity of the blood and tissue fluids.

And if we get these results in a natural physiological condition, how much more shall we get them in a condition of disease such as acute rheumatism! The patient is in bed, and the limbs so painful that he cannot move a muscle; he has little or no appetite, and what food he does take is imperfectly digested owing to the effect of the fever on the digestive organs; it is little wonder, then, that his urea and acidity run rapidly down and soon become very low indeed, and though the fever no doubt keeps them up for a time, it soon has insufficient supplies to work upon, and begins to lose its power.

This action of the skin in lowering acidity is clearly seen in Dr. Wilks' case mentioned above, where it is noted that the patient perspires freely, and five days later the note says that his urine is alkaline and his pains are gone.

I have no doubt that the alkalinity of the urine was due to the causes I have attempted to outline above, and that when Nature took the matter in hand she acted so thoroughly that a small dose of acid had very little effect; and I know from experience that when a patient is run down by exhausting disease it is not easy to raise the acidity of the urine very much by giving acids.

While speaking of skin activity and its effect on the acidity of the urine I should like to quote what Sir A. Garrod has said on this subject, as I believe it to be a point

the importance of which has not as yet been thoroughly realised. Thus, in his work on 'Gout and Rheumatic Gout,' 3rd ed., p. 258, he says, "Suppressed perspiration is immediately followed by an increase of urinary acidity;" and I can not only amply confirm this assertion, but can show also that the converse is true, and that increased perspiration lowers the acidity of the urine.

Let us look for a moment at what is said by some recognised authorities on the question of the skin excretion, especially the perspiration; thus Besnier ('Dictionnaire des Sciences Médicales,' p. 496) says, "Dans le rhumatisme articulaire aigu comme dans toutes les affections sudorales la sueur examinée au moment de sa production sur un surface de la peau convenablement debarrassé par le lavage des enduits sébacés et des produits de décomposition de l'épithelium et des corps gras, la sueur est à peu près neutre dans le plus grand nombre de cas, aussitôt qu'il s'est établi une véritable diaphorèse, plus nettement acide quand elle est peu abondante ou qu'elle commence à couler, exceptionellement alcaline."

Sir A. Garrod (Reynolds' 'System,' vol. i, p. 896) says, "The perspiration is generally considered to be intensely acid in acute rheumatism; in several cases I have found it less acid than in healthy subjects; but it must be remembered that the amount of perspiration is excessive."

Prof. M. Foster ('Physiology,' 5th ed., part ii, p. 695) says, "When sweat is scanty the reaction is generally acid, but when abundant it is alkaline, and when a portion of the skin is well washed the sweat which is collected immediately afterwards is usually alkaline."

With regard to the above quotations my friend Dr. A. E. Garrod tells me that he has himself seen several cases of acute rheumatism where even in the early stages of the disease the perspiration collected after the surface of the skin has undergone careful cleansing has been neutral or even alkaline, so that he is quite prepared to endorse Besnier's observations just quoted.

With regard to this point I would remark, firstly, that

the perspiration must be moderately copious if it is to be collected at all in this way—a fact, it will be observed, very properly pointed out by Sir A. Garrod in the above quotation from Reynolds' 'System;' and secondly, that it does not follow that a very large amount of acid is not got rid of by the skin in the twenty-four hours because the excretion is neutral or even alkaline for a short time, and when very copious. The urine is often alkaline for a few hours in the morning, but yet the acid excreted in the whole day may be considerable; and again, when the urine is very copious the acidity may appear very low till we come to multiply the acidity by the quantity excreted.

But the fact of most consequence, and which to my mind absolutely deprives the above line of argument of all force as regards the effect of the skin excretion on the reaction of the blood and tissue fluids, is the one above mentioned as pointed out by Sir A. Garrod, viz. that suppression of perspiration is immediately followed by a rise of urinary acidity; and the further fact which I can vouch for, and which it is easy to demonstrate, that increased skin action and perspiration are followed by a fall in urinary acidity. I take it, therefore, that in spite of the above observations on the reactions of the perspiration a very considerable amount of acid is got rid of in the excretions of the skin both in health and disease, and that this is often enough to depress very considerably the acidity of the urine, and raise to a corresponding extent the alkalinity of the blood and tissue fluids.

It seems, then, that all the methods of treating acute rheumatism that are of any value have one effect in common, the causation of a *plus* excretion or elimination of uric acid.

And further, that as regards salicin and compounds of salicylic acid, their utility in the disease is directly proportional to their power of eliminating uric acid.

May we then go further, and say that acute rheumatism is due to uric acid? I for one should be inclined to say

that the joint pains of this disease are undoubtedly due to uric acid; but there remains still the question, what drives the uric acid into the joints?

To this I would reply, high and rising acidity of the urine and concomitant decreased alkalinity of blood and tissue fluids, acting on uric acid in this disease just as it does in gout. But why then is rheumatic fever so different in many ways from gout?

I do not know that I can completely answer this question, but I have a very strong impression that it may be due to a difference in the amount of uric acid present, and to a difference in the activity of metabolism of young subjects in whom rheumatic fever occurs, as compared with that of older subjects in whom gout is met with. The chief difference is a greater activity in the metabolic processes of the young, for while an adult forms and excretes some three or four grains of urea per pound of body-weight, a child of three or four years may excrete as much as nine or ten grains per pound, as I can show from my own investigations; and with this larger formation of urea in a child there is a greater formation of uric acid, and, what is perhaps more important, a greater formation of acids. It thus appears evident that a child might soon have much more uric acid in its blood than an adult could easily get, and any little febrile disturbance might raise the acidity very greatly and precipitate the uric acid into the joints.

Of course it is quite possible that an essential factor in acute rheumatism is the formation of an acid in large quantities by some fermentation process, or, as has been suggested, by a bacterium ('Brit. Med. Journ.,' i, 1887, p. 1381). I cannot express any opinion on these points, but I do believe very strongly that the essential feature of acute rheumatism is the retention of uric acid in the joints and tissues of the body by a high and rising acidity, and that this, and this only, will enable us to explain completely the results obtained with drugs and diet in the treatment of this disease.

Sir A. Garrod has recorded the fact[1] that he has repeatedly examined the blood in acute rheumatism, and has never been able to find any uric acid there; he says, "The absence of uric acid or urate of soda is important, as it at once shows an essential difference between gout and rheumatism." But this is exactly what I should expect, for it is not likely that the uric acid can be in two places at once; and if, as I am supposing, a high and rising acidity has driven it all into the joints, it is not likely that any will be found in the blood. And some of Sir A. Garrod's own observations lend, I think, strong support to this explanation, for he has pointed out ('Gout and Rheumatic Gout,' pp. 187 and 274) that there is no uric acid in the fluid of a blister, or in blood drawn directly over the inflamed joint in gout; and he proceeds to argue from these facts that inflammation destroys uric acid.

It has, however, been shown that fever[2] lowers the alkalinity of the blood and raises the acidity of the urine. This would, as I have shown ('Journal of Physiology,' vol. viii), cause a diminished excretion of uric acid in the urine, and, as I have argued, diminish also the amount of it in the blood; and there can be very little doubt that a local inflammation will have the same effect, viz. to diminish the alkalinity of the blood and tissue fluids, and drive the uric acid they contain out of solution. So that while I think that Sir A. Garrod's facts are perfectly correct, I believe that the result he notices is due to a precipitation of uric acid, and not to a destruction of it.

This explanation affords us also an insight into the causation of one of the differences between gout and rheumatism; for I believe I am correct in saying that the temperature in acute rheumatism is generally considerably higher than in gout, and therefore the effect on the alkalinity of the blood and the precipitation of urates from solution will be more complete in the former than in the latter disease.

[1] Reynolds' 'System of Medicine,' vol. i, p. 897.

[2] Dr. Peiper, 'Virchow's Arch.,' June, 1889, p. 337.

There has been recently published ('Brit. Med. Journ.,' i, 1890, p. 472) a paper by Dr. B. N. Dalton on the "Ætiology of Rheumatic Fever," in which he urges that this disease may be "caused by breathing air contaminated by the emanations from sewers and drains," and gives many interesting facts and cases in support of his argument.

From my point of view such a mode of causation is extremely probable, for if the sewer emanations give rise to fever (as there is no difficulty in believing that they may do), they will, as we have seen, raise the acidity of the urine, and diminish the alkalinity of the blood and tissue fluids; and under certain conditions of metabolism which are often present in young subjects they may cause the precipitation of a large amount of uric acid in the fluids and tissues of the joints, thus producing what is known as rheumatic fever.

The way in which the uric acid is precipitated on the fluids and tissues of joints I have pointed out in previous papers (see 'Brit. Med. Journ.,' ii, 1888, p. 12).

I have also pointed out that Sir A. Garrod has shown that the tissues and fluids of the joints are less alkaline than the tissues and fluids of the body generally; in this respect they resemble the spleen, in which uric acid is constantly found, so that the precipitation in the spleen and in the joints stands on the same ground, and is supported by the same facts and argument.

In a paper in Wood's 'Medical and Surgical Monographs,' New York, February, 1890, I have suggested that some fevers may act on uric acid in exactly the same way, and thus produce rheumatic fever; and also that tonsillitis and even some local inflammations, as an alveolar abscess, of which a case is narrated, may by raising the temperature, and so the acidity, produce the same result.

Though I speak here mostly of the effects of fever I do not wish to lose sight of the fact that several other causes may raise the acidity, as, for instance, suppression of perspiration, as pointed out by Sir A. Garrod; and a severe

chill is a commonly accredited cause of rheumatic fever. Another is the ingestion of acids and acid-forming foods; and in this way I believe it is possible to produce "rheumatic" (uric acid) pains in almost anyone. Indeed, I have often produced such pains in patients, the subjects of high arterial tension, when giving them acids, opium, and other drugs to reduce their tension.

Again, acids may be formed in the stomach to a very considerable extent, as pointed out by Bouchard, and I am sure from my own experiences that gastro-intestinal troubles have a most important connection with some cases of rheumatism.

Moreover, as previously mentioned, it has been suggested that some fermentation processes, with or without the agency of a bacterium, may produce considerable quantities of acid.

The excretion of uric acid in rheumatic fever is enormous, and only to mention one case I found upwards of 26 grs. in the urine of a man with this disease on the first day of taking salicylate of soda, having a relation to urea of 1 : 17, the normal relation being 1 : 33. There was here double the ordinary quantity of uric acid per grain of urea; and if the additional 13 grs. of uric acid were all in the joints, it would, I think, account for most of the trouble they gave. For I can cause very distinct symptoms in my own joints by precipitating only 2 or 3 grs. into them.

Again, when salicylate is left off after a rheumatic attack there is often very marked high arterial tension to be observed for some days, thus pointing, I believe (see paper on "Uric Acid and Arterial Tension," 'Brit. Med. Journ.,' i, 1889, p. 288), to excess of uric acid combined with alkali in the blood; and if at this time fever, dyspepsia, or excess of nitrogenous food causes a sharp rise in acidity, a relapse is the common and easily explained result.

It would lead me too far to go minutely into the action of all these causes here, but in conclusion I will merely suggest—

1. That the essential feature of rheumatic fever (viz. the joint symptoms) is the result of a precipitation or concentration of all, or nearly all, the uric acid in the body, in the tissues and fluids of the joints.

2. That this concentration is due, as in gout, to high and rising acidity or greatly reduced alkalinity of the tissue fluids, of which the high acidity of the urine may be taken as an index, or the reaction of the blood may be taken as more direct evidence.

3. That the completeness of this precipitation accounts for the absence of uric acid from the blood in rheumatic fever, as noticed by Sir A. Garrod.

4. That such a process of causation enables us to explain completely the action both of drugs and diet in rheumatic fever; and, lastly—

5. That, as it has been the object of this paper to point out, the compounds of salicin and salicylic acid have a curative power in rheumatic fever which is precisely proportional to their powers of eliminating uric acid, and that they cure the disease by effecting such elimination.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 105.)

ON THE CONDITION OF THE REFLEXES

IN CASES OF

INJURY TO THE SPINAL CORD;

WITH SPECIAL REFERENCE TO THE INDICATIONS FOR OPERATIVE INTERFERENCE.

BY

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Received March 25th—Read May 13th, 1890.

On February 25th of the present year a paper was read before this Society by Dr. Charlton Bastian on the "Symptomatology of Total Transverse Lesions of the Spinal Cord, with special reference to the Condition of the various Reflexes." The author pointed out that, contrary to the generally accepted teaching, the deep reflexes were abolished in such cases; and he supported his contention by cases observed by himself, and by reference to the observations of others.

In the discussion which ensued I ventured to speak of my own experience of injuries of the spinal cord, and stated

that in twenty-two cases observed during the past six years at St. Bartholomew's Hospital the reflexes were never exaggerated when the spinal cord was completely crushed. Since speaking on this subject it has seemed to me that, considering the importance of the issues raised, it would be advisable to record somewhat more in detail the cases I have observed, and I am encouraged to do so by the fact that in but few published examples of injury to the spinal cord is there any definite statement of the conditions of the reflexes.

I am aware that the opinions expressed in this paper are at variance with the accepted doctrines of physiology and with the results of experiments, but the facts I record can be supported by the testimony of various independent observers who have seen the patients with me, and I have in all the cases demonstrated the conditions of the reflexes to the students dressing in the surgical wards of St. Bartholomew's Hospital. Microscopical preparations of the spinal cords were made in most cases, and for several of them I am indebted to my friend Dr. Howard Tooth.

For the purposes of this paper I have taken twenty-one cases of complete crushing of the spinal cord, in all but one of which this condition was confirmed by post-mortem examination. In ten cases, however, the patients survived but a few hours, and I have not therefore thought it advisable to make use of them in considering the state of the reflexes, although I have alluded to the conditions found at the autopsies in considering the question of operative interference. I may state, however, that in no single case were any reflexes or rigidity of muscles shown, although, on the other hand, in the more rapidly fatal cases they were not examined for by those who treated them, and such patients were not seen by myself.

In the eleven other cases, notes of which are appended, the various reflexes were especially observed by myself as well as by others, and records were made at the time.

I have also given brief notes of cases of injury of the spinal cord when from the symptoms it was evident that

there had not been complete solution of continuity, and have pointed out that in these the reflexes are increased. I may further add in explanation of the term "fracture dislocation" which is frequently employed, that by it is implied a partial dislocation of the body of one vertebra from that of another, combined with fracture of the body or processes of the displaced bones.

Cases of Complete Transverse Lesions of the Spinal Cord.

CASE 1.—T. B—, æt. 45, fell through a window on to his head on August 7th, 1888. On admission under Mr. Willett he complained of pain in his neck and of loss of power and of sensation in the trunk and limbs. He was found to be completely paralysed in the lower extremities and the trunk. There was anæsthesia of the abdomen, and of the thorax below the nipples. Respiration was almost entirely diaphragmatic. There was incontinence of fæces and retention of urine. The arms were not paralysed.

9th.—Priapism was marked.

10th.—Reflexes examined. The legs and thighs are flaccid; there is no clonus or rigidity; the knee-jerks are absent on each side; there is no reflex on tickling the feet.

The patient developed cystitis and bronchitis, and died on September 23rd, the reflexes not having returned.

Post-mortem examination showed a fracture dislocation at the second and third dorsal vertebræ. There was no pressure on the cord, and the membranes were uninjured. The spinal cord itself appeared natural until it was cut open; its structure then appeared blurred and homogeneous, and a microscopic examination showed below the seat of fracture the usual typical descending degenerations.

CASE 2.—John H—, æt. 58, fell from a height on to his head on July 17th, 1887, and was at once paralysed. He was admitted into Colston Ward under Mr. Langton,

and on July 23rd there was found to be complete anæsthesia of the lower extremities, of the abdomen, and of the thorax below the second intercostal space. The respiration was diaphragmatic. The sphincter of the rectum was paralysed. The urine was retained. The extensors of the forearms were paralysed, but the elbow, fingers, and wrists could be flexed. There was no rigidity, no ankle-clonus, and no knee-jerks. There was slight sole reflex on each side, but no cremasteric reflex.

Three days later the patient died.

A post-mortem examination showed a fracture dislocation of the fifth and sixth cervical vertebræ, the fragments being in good position and not compressing the cord. The membranes were untorn, but the cord was completely crushed to a pulp.

Case 3.—A man, æt. 42, fell on the back of his head on October 17th, 1887, and at once lost power in his lower extremities. He was admitted into Colston Ward under Mr. Langton. The day after the injury his lower extremities were found to be completely paralysed, the abdominal and thoracic muscles were paralysed, and sensation was lost below the third rib in front. There was incontinence of fæces and retention of urine. There was but little anæsthesia of the arms, but the extensors of the forearms, wrists, and fingers were paralysed. There was slight priapism.

On October 21st the reflexes were examined. Both deep and superficial reflexes were completely lost in the lower extremities. There was no rigidity.

November 7th.—On tickling the soles there was decided movement of the toes. No clonus or rigidity.

The patient died a week later, and a post-mortem examination showed a fracture dislocation of the sixth and seventh cervical vertebræ, without any displacement. The spinal cord was quite pulped.

Case 4.—A lad, æt. 18, fell down a lift on June 17th, 1887, and was admitted into Rahere Ward, under the care

of Mr. Baker. The same day he was found to have complete paraplegia with retention of urine and incontinence of fæces. Respiration was diaphragmatic, and sensation was lost below the nipple. The extensors of the forearms and wrists were paralysed.

On June 20th there was found to be some further loss of sensation, so that now anæsthesia was complete below the second rib. Priapism was marked, and was increased by catheterisation.

29th.—Examined for reflexes. There was no rigidity of the lower extremities, and both superficial and deep reflexes were absent.

November 1st.—Still no reflex in lower extremities.

December 1st (twenty-four weeks after the accident).—Distinct return of the sole reflexes, the toes being moved when the foot is tickled. No patellar reflex; no clonus; no pectoral reflex. The patient says that he has a tingling and pricking sensation over the chest when a catheter is passed, and after its passage there is profuse perspiration over the head, face, and neck, and the development of a bright red rash which persists for fifteen or twenty minutes.

31st.—No change in the reflexes.

January 26th, 1888.—The patient died.

A post-mortem examination showed a fracture dislocation at the junction of the seventh cervical and first dorsal vertebræ. There was no displacement at the time of death. The membranes were intact, but the spinal cord was crushed to a pulp.

CASE 5.—H. W—, æt. 54, fell from a height of sixteen feet on to a wall, striking his back. He at once felt as though he had lost his legs.

He was admitted under the care of Mr. Morrant Baker into Harley Ward, where two days later, *i. e.* on September 11th, 1884, his breathing was found to be chiefly diaphragmatic, and his abdominal muscles and lower extremities to be completely paralysed. His urine was retained, and

motions were passed involuntarily. His legs and thighs were quite limp and flaccid, and there were no knee-jerks or ankle-clonus on either side. The cremasteric, sole, and epigastric reflexes were also absent.

The patient gradually sank, and died on January 30th, 1885, from suppurative nephritis, having survived the accident nearly six months.

A post-mortem examination showed that the bodies of the fifth and sixth dorsal vertebræ had been fractured. The fracture had united, but there was no compression of the cord. The dura mater was a little thickened, but was not torn open.

The spinal cord looked as though it had been pinched opposite to the seat of fracture, and was here quite diffluent. Microscopical examination showed that it was completely disorganised.[1]

Case 6.—Mary C—, æt. 54, fell downstairs on November 17th, 1886, and was found lying on her back in a helpless condition.

She was brought to St. Bartholomew's Hospital, and was admitted into President Ward under the care of Mr. Willett. She had severe pain in the neck; the legs, thighs, abdomen, and thorax were anæsthetic; the respiration was diaphragmatic; there was complete loss of power in all the muscles of the lower extremities; the sphincter ani was paralysed, and the urine was retained. The upper extremities were partially paralysed, the pectorals, deltoids, and biceps alone acting. The hands and forearms were nearly quite anæsthetic. The lower extremities were flaccid, and the knee-jerks and the sole reflexes were absent. The patient died on November 20th, three days after the injury, without there having been any alteration in the symptoms. A post-mortem examination showed a fracture of the fifth and sixth cervical vertebræ with complete crushing of the cord. The cord was not compressed by displacement of the fractured vertebræ.

[1] See report by Dr. Tooth in 'St. Bartholomew's Hospital Reports,' vol. xxi, p. 141.

CASE 7.—Florence S—, æt. 31, fell on her head on July 11th, 1885, and was at once paralysed. She was admitted into Lawrence Ward under the care of Mr. Smith. There was complete paraplegia with incontinence of fæces and diaphragmatic breathing. Sensation was lost on the inner side of the arms, and the extensors of the forearms and wrists were paralysed. There was no rigidity, and the reflexes of the lower extremities were lost. The patient died in four days, and a post-mortem examination showed a fracture dislocation of the fourth and fifth cervical vertebræ with complete crushing of the spinal cord. There was no pressure on the cord by displaced fragments, and the dura mater was not torn.

CASE 8.—Henry S—, æt. 41, fell from a height of twelve feet on to his head, and was admitted into hospital under Mr. Baker in an unconscious condition on March 14th, 1889. He soon regained consciousness, and then complained of great pain in the neck, and was found to be completely paraplegic. A more complete examination next day showed that there was complete anæsthesia below the level of the second rib, and paralysis of the muscles of the chest, abdomen, and lower extremities. Urine was retained and fæces were passed involuntarily. In the upper extremities there was numbness in the distribution of the ulnar and internal cutaneous nerves. The lower extremities were quite flaccid, and the sole reflex and the knee-jerk were absent on each side. There was no clonus. The patient survived a week, but the reflexes did not return. A post-mortem examination showed a fracture dislocation of the sixth and seventh cervical vertebræ without any material displacement of fragments. The dura mater was intact, but the spinal cord was completely crushed.

CASE 9.—A man, æt. 63, fell from a scaffolding on to some rails on December 6th, 1884, and was at once paralysed. On admission into St. Bartholomew's Hospital under Mr. Baker he was found to have complete loss of

motions were passed involuntarily. H[illegible]ond rib, with paresis were quite limp and flaccid, and ther[illegible] and sole reflexes were or ankle-clonus on either side. [illegible]jerks. The limbs were and epigastric reflexes were also [illegible] day after the accident, and

The patient gradually sank [illegible] showed a fracture dislocation 1885, from suppurative ne[illegible] vertebræ with laceration of accident nearly six month

A post-mortem exam[illegible]

the fifth and sixth d[illegible] æt. 43, fell off a ladder on April 12th, The fracture had ur[illegible] into Henry Ward under Mr. Smith the cord. The d[illegible] complete loss of sensation and motion not torn open. [illegible]ities, with loss of sensation below the

The spinal [illegible]rib, and paralysis of the muscles of the opposite to [illegible] as well as of the lower intercostals. He diffluent. [illegible] great pain in the back. The urine was re- complete[illegible] [illegible]here was marked priapism; there was also [illegible] of faces. Further examination showed that

Cas[illegible] [illegible]xtremities were quite flaccid, and that the sole, 17th, [illegible] and patellar tendon-reflexes were all absent. con[illegible] [illegible] was no clonus. As there was no change in the [illegible] of the patient he was sent to an infirmary ten w[illegible]

[illegible] II.—W. G—, a man æt. 52, was admitted into [illegible] Ward under the care of Mr. Langton on March [illegible] He had fallen from a scaffold, and was picked [illegible]. The day after admission, when he had [illegible] consciousness, I found him quite paraplegic, [illegible] absolute anæsthesia and loss of power below the third [illegible] there was marked priapism. The hands and arms [illegible] feeble, but there was no definite paralysis or loss of [illegible]. Breathing was difficult. An examination of [illegible] reflexes showed complete loss of patellar tendon- [illegible] with absence of clonus and rigidity. The cremas- [illegible] reflexes were absent, but touching the skin of the [illegible] caused increased priapism. There were no sole [illegible]exes.

The following day I examined him again with Dr. [illegible]mrod. The right sole reflex had returned, but the

deep reflexes were unaltered. The contraction of all the muscles of the lower extremities to a direct blow was much ceased. The supinator reflex on the right forearm was sed.

March 15th the left sole reflex had returned, and of anæsthesia had extended as high as the second

On March 17th the patient died of congestive pneumonia without further alteration in the reflexes.

A post-mortem examination showed a fracture dislocation of the first and second dorsal vertebræ. The dura mater was untorn, but the spinal cord was completely crushed, and was quite diffluent.

Cases of Partial Lesion of the Spinal Cord.

Case 1.—J. R—, æt. 16, was admitted into Harley Ward under Mr. Morrant Baker on June 6th, 1889, having fallen from a height of six feet on to the back of his head.

On admission he was found to be conscious, but could not nod his head or rotate it on account of pain in his neck. No deformity could be seen. The patient complained of "numbness all over," but was not completely paralysed, although unable to stand. Both legs could be moved, but the left leg was very feeble, and neither limb could be raised from off the bed. Both knee-jerks were increased. The next day he was more carefully examined, and it was found that he could move the right leg feebly, although the left was quite paralysed. The intercostal muscles acted feebly. Urine was retained, but the sphincter ani was not relaxed. The hands and forearms were not paralysed, but were very weak.

June 8th.—Can move both legs a little, and has passed his water naturally.

11th.—Has gradually gone back again, and has retention of urine and further loss of power in the left leg.

sensation and of motion below the second rib, with paresis of the left arm. The epigastric and sole reflexes were absent, as were also the knee-jerks. The limbs were flaccid. The patient died the day after the accident, and a post-mortem examination showed a fracture dislocation of the fifth and sixth cervical vertebræ with laceration of the spinal cord.

Case 10.—A man, æt. 43, fell off a ladder on April 12th, 1888. On admission into Henry Ward under Mr. Smith he was found to have complete loss of sensation and motion in the lower extremities, with loss of sensation below the level of the fifth rib, and paralysis of the muscles of the abdominal wall as well as of the lower intercostals. He complained of great pain in the back. The urine was retained, but there was marked priapism; there was also incontinence of fæces. Further examination showed that the lower extremities were quite flaccid, and that the sole, epigastric, and patellar tendon-reflexes were all absent. There was no clonus. As there was no change in the condition of the patient he was sent to an infirmary ten days later.

Case 11.—W. G—, a man æt. 52, was admitted into Colston Ward under the care of Mr. Langton on March 12th, 1890. He had fallen from a scaffold, and was picked up unconscious. The day after admission, when he had recovered consciousness, I found him quite paraplegic, with absolute anæsthesia and loss of power below the third rib. There was marked priapism. The hands and arms were feeble, but there was no definite paralysis or loss of sensation. Breathing was difficult. An examination of the reflexes showed complete loss of patellar tendon-reflex with absence of clonus and rigidity. The cremasteric reflexes were absent, but touching the skin of the penis caused increased priapism. There were no sole reflexes.

The following day I examined him again with Dr. Ormerod. The right sole reflex had returned, but the

deep reflexes were unaltered. The contraction of all the muscles of the lower extremities to a direct blow was much increased. The supinator reflex on the right forearm was increased.

On March 15th the left sole reflex had returned, and the area of anæsthesia had extended as high as the second rib.

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June 8th.—Can move both legs a little, and has passed his water naturally.

11th.—Has gradually gone back again, and has retention of urine and further loss of power in the left leg.

The left knee-jerk is absent, but the right knee-jerk is increased.

13th.—Better again. Both knee-jerks are increased. He has more power in the legs and in the arms. Passes urine normally.

20th.—Some rigidity of both lower extremities with exaggerated knee-jerks and clonus. The left arm and leg are very weak, and can scarcely be moved. The right arm is fairly strong, but the right leg is very feeble. Micturates naturally.

July 25th.—Has slowly improved in every way, but has a good deal of pain in the neck.

August 29th.—Continues to improve. Sensation has returned considerably in both upper and lower extremities, and there is definite increase of power in the left arm and leg.

October 3rd.—Going on well. Patellar reflexes still exaggerated, and some tremor in quadriceps extensors. Ankle-clonus well marked. Sole reflex increased. Lower extremities still a little rigid. Power of movement much improved.

24th.—Knee-jerks not so much exaggerated. Is much better in every way, and gets out of bed in the evening.

November 21st.—Can walk with the aid of a stick.

29th.—Discharged. Left leg and arm weak. Reflexes of lower extremities still exaggerated.

February 20th, 1890.—Almost quite well, but complains of some remaining weakness with left arm, although this is rapidly improving. Knee-jerks are still exaggerated, although but slightly.

Case 2.—T. L—, æt. 39, fell off a van on April 19th, 1884, and injured his back. He was admitted into Colston Ward under Mr. Langton. His legs felt numb and powerless directly after the injury, and on examination at St. Bartholomew's he was found to be almost quite paraplegic.

Next day his reflexes were examined. The lower ex-

tremities were rigid, the patellar reflexes were increased, and tickling the soles caused spasmodic twitching of the muscles of the thigh and leg.

The patient quickly improved, and in three days' time he was again able to move the legs and thighs. A week later he could get out of bed and walk with the aid of crutches.

Numbness, rigidity, and increased reflexes continued for some months.

CASE 3.—A man, æt. 28, fell and struck his spine, and at once felt a loss of power and numbness in his lower extremities. He was admitted into Abernethy Ward under the care of Mr. Savory, and was found to have partial paralysis of the legs and thighs, but no incontinence of fæces or retention of urine. He could lift the legs off the bed whilst lying down.

Next day further examination showed increase of knee-jerks on both sides, muscular tremor and spasm on tickling the soles, and ankle-clonus. He rapidly improved, and made a complete recovery in fourteen days.

I have purposely abstained from entering into any lengthy details as to the course and complications of the cases here recorded, as the object of this paper is to direct attention to a few definite observations. The first and chief fact which is demonstrated is that in cases where the spinal cord is completely crushed in the cervical or dorsal regions the deep reflexes are at once lost and do not return. I am aware that it has been formerly suggested as an explanation of this that the cause of their disappearance is shock, but this theory has never been supported by anything in the shape of proof. On the other hand, it is clearly shown by the cases here described that after the lapse of a time much more than sufficient to allow of recovery from shock the deep reflexes are absent. Thus one patient survived a month, another six weeks, a third five months, and a fourth ten months.

The superficial reflexes are also generally lost immediately after the accident, although this is not invariably the case, and they, unlike the deep reflexes, may in time return. In Case 2 the superficial reflexes were never lost. In Case 3 they returned on the eighteenth day, but in Case 4 not until the twenty-second week. In Case 5, when the patient survived five months, they were not noticed to return, and this also happened in all the other patients who survived for periods varying from one day to six weeks, with the exception of Case 11, in which the sole reflexes returned on the second and third days following the injury.

On the other hand, when the cord has been injured, and when it is compressed, but when also its continuity has not been entirely interrupted, the reflexes are not only preserved, but may be, and generally are exaggerated.

The bearing of these facts on the question of operation is obvious. If the limbs are flaccid and reflexes are absent the diagnosis is that the cord is completely severed; and, as it is known that in the human subject such a lesion is never repaired, operative interference is useless, and should not be undertaken. If, however, in spite of paralysis more or less complete there is rigidity and increase of reflexes, then the diagnosis is that there is but partial severance of the cord, and if there be any indications for operation, such as apparent compression by displaced bone, there is justification for such a measure.

It must, however, be pointed out that operative interference can but seldom be of avail, and that in the vast majority of cases of fracture dislocation of the spinal column no good whatever can arise from it. This is made abundantly clear by a consideration of the conditions found on post-mortem examination. Among the ten fatal cases I have recorded where the spinal cord was found, post mortem, to have been completely crushed, there was no pressure by displaced bone in any one. In addition to these I have made post-mortem examinations of nine other examples of fracture dislocation, and of one

of simple dislocation without fracture, and in not one of them was there found any compression of the cord by displaced bone. From a consideration of the conditions found on post-mortem examination of these twenty cases I think it may be concluded that the cord is injured by a forward dislocation of the body of the upper of the two vertebræ involved in the injury, and that the spinal cord is suddenly and violently stretched and crushed across the upper and posterior margin of the body of the vertebra below. As soon as the force which has caused the injury is withdrawn the displaced vertebra is restored partially or entirely to its natural position by the elasticity of the ligaments and the contraction of the muscles. Unfortunately, however, the mischief is already done, and the cord is injured beyond repair. In most of the cases I examined I found the dura mater uninjured, and in many of them the cord within it at first appeared natural. It was often only on section that the amount of injury inflicted could be estimated.

In conclusion I would point out that although I consider there is sufficient proof that in all cases of total transverse lesion of the spinal cord the deep reflexes are abolished, and that in cases of partial lesion the reflexes are increased, I am not prepared to assert definitely that in all cases of partial lesion there is necessarily such increase. It may be that in some severe cases of this class the reflexes are also abolished for a time, and I am acquainted with a case, which I did not myself observe, in which in a patient with undoubted partial lesion the reflexes were said to be abolished.

This is a matter which may easily be determined by future careful examination.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 114.)

SENILE HYPERTROPHY AND SENILE ATROPHY OF THE SKULL.

BY

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I AM desirous of directing the attention of the Society, in a more especial manner than has hitherto been done, to two changes of opposite nature which are not very infrequent in the skulls of elderly persons, affecting chiefly the vault of the skull, both of which, so far as I know, are peculiar to this part of the skeleton, and for neither of which is it easy to offer a thoroughly satisfactory explanation. One of these consists in an increase of bony material and weight—a hypertrophy; the other in a diminution of bony material and weight—an atrophy.

It is well known that shrinkage of the brain-substance, associated with old age, general wasting or prolonged alcoholism, is commonly attended with an increase of fluid in the meshes of the pia mater or a thickening of the calvarial part of the skull or with both these conditions. Both conditions, we may judge, proceed from the same cause, viz. a lessening of the pressure in the cranial cavity resulting from the brain-shrinkage, a consequent dilatation of vessels and a slowing of circulation in them which leads to an increased filtration of their contents, serous and cellu-

lar, and a dropsy of the pia mater or a hypertrophy of the calvaria or both. The condition is comparable with that of a part under a bell-glass (or cupping-glass) or other exhausted receiver; and the results are similar to those which take place in chronic œdema, viz. an increase not only of the fluid or serous components of the blood but of the cell-constituents also, and an increase of tissue-development. The latter, indeed, is not an uncommon sequence of prolonged dropsical condition from whatever cause it arises. Thus prolonged œdema of the lower limbs is often attended with thickening of the connective tissues, as illustrated in elephantiasis arabum, where the hyperplasia may extend to the bones causing thickening of them with nodular outgrowths; and thickening and sclerosis of bones readily follows upon congestion of blood-vessels induced by various causes, that is to say, the greater or less nutrition of these tissues, of bone in particular,—their hypertrophy or atrophy—is dependent, partly, upon their own nutritive energies, partly, upon the greater or less supply of nutritive material and tissue-forming elements, and, partly, upon the more or less true balance between these two. And it may be, or rather must be, a feature of proper and properly regulated nutritive force to appropriate the required nutritive material and no more—not merely to turn to account, but to control and keep within bounds, the energies of the leucocytes with which they are supplied. The paradox may thus come about that in the connective tissues, more particularly bone and fat, under certain circumstances, a decrease of nutritive force may lead to an increase of nutrition or hypertrophy; and it may be that the same failure of nutrition which in the aged person causes wasting or atrophy of the highly organised brain-tissue may cause thickening or hypertrophy of the more lowly organised brain-case, first, by inducing an increased afflux of blood there, and, secondly, by disabling the osseous tissue of the skull from controlling the ossifying tendencies of the blood-elements effused into it.

Whatever may be the view of the precise pathology of

the condition, it seems to have its analogue in the enlargement of the prostate and the thickening of the arteries so common in old people.

The coloured drawings (Pl. III, and Pl. IV, figs. 1 and 2) show the congested state of the diploë and of the inner tables of the skull-vault from an alcoholic man, aged 50, who died in Addenbrooke's Hospital of apoplexy in the early part of this year. The skull-wall is somewhat thickened by bone deposit on the interior, and is slightly denser than natural. I have lately seen a similar, though not so marked, congestive condition in the skull of a man aged 73; and the large size of the vascular canals often seen on the interior of the senile skull renders it probable that a state of congestion is not uncommon in the vessels of this part in those who are advanced in life.

The thickening of the skull-wall in old people takes place chiefly, if not exclusively, on the interior, and is commonly first and most marked beneath the domes of the frontal bone, on the two sides of the median line, over the part, that is, of the frontal lobes where brain-shrinkage is first and most marked. It alters the inner contour of these domes, flattening them, or even causing them to bulge, on the interior. After a time it affects the whole of the frontal bones and the rest of the vault, and may reach the base, thus extending over all the interior of the skull. In some cases the frontal and parietal bones thus thickened are cancellous, the diploë being increased and advancing upon the receding inner table, and there may be little or no increase of weight. In other cases, and more frequently, the inner table is thickened and bony deposit takes place in the diploë, which is thereby condensed; and the skull-wall is not only thickened but rendered throughout denser and heavier. In some the condensation or obliteration of the diploë has taken place without much thickening of the skull-wall. It is the increase at the expense of the cranial cavity which distinguishes the thickening of the skull in old age and in other cases of brain-shrinkage from the thickening that

takes place in osteitis deformans and some other low inflammatory affections, for in these the increase is caused by addition to the *exterior*.

The following specimens in the Cambridge Pathological Museum illustrate these points :—1. A nearly edentulous skull with great thickening of the wall and increase of the diploë except at the base, the thickening being at the expense of the cranial cavity. In this case there is not much increase of weight. 2. An edentulous skull without lower jaw and with atrophied superior maxillary and facial bones, but with thick dense skull-wall, weighs 34 oz. 3. The edentulous skull of an aged female with much wasted maxillary and facial bones, weighs 24½ oz. The bones of the cranium are not thick but rather dense, and the ridges in the interior are somewhat pronounced. 4, 5, 6. Three skulls without lower maxillæ, edentulous, and with the usual thinning of the superior maxillary and facial bones, weigh respectively 28½ oz., 28 oz., and 26½ oz. 7. The skull of a man reputed to have died at 104 from which the lower jaw and all the back part (about a quarter of the whole cranium) has been removed, and which is edentulous and with wasted facial bones, weighs 17 oz. 8. The lower part of an edentulous and evidently very aged skull from which the upper part has been removed a little above the orbits, weighs 15 oz. 9. A thick dense piece of the upper part of the skull from a woman aged 80. In all of them, except No. 1, the bones are dense and more or less thickened ; there has been addition of osseous matter interstitially as well as upon the inner surface ; and the contrast between the thick, heavy, dense cranium and the thin light facial bones is marked in all these instances. I have long been in the habit of illustrating this as well as the contrast with the other bones of the skeleton by showing the skull and thigh-bone which I took from a woman reputed to have died at 103, and which are in the same museum. Although only one tooth remains, the alveolary processes are nearly gone, and though the maxillary and other facial bones are thin and

light, yet the skull weighs 28½ oz., which is above the ordinary weight of the adult skull in which the teeth remain. The increase of weight is due to the thickness and density of the cranial bones, the tables being thick and the diploë dense. The encroachment upon the cranial cavity is, as usual, most marked under the frontal domes, but there has been some deposit upon the whole of the interior. The thigh-bone of this person, though large and well formed, weighs only 5 oz.; the reduction of weight being caused by absorption of the cancelli and thinning of the bone-wall from the interior. The other bones of the skeleton were in a similar atrophied condition; and the want of correspondence between the thick, heavy skull and its fragile supporters was very striking. It should be said that the old woman had latterly been bedridden.

The problem of the cause of the ill-assorted condition of these bones—the dense heavy skull and the light porous fragile thigh-bones in the same person—is not very easy to solve. The increase and density of weight in old people is, so far as I know, quite peculiar to the skull-wall. All the other bones, as age advances, become lighter and undergo absorption, which commences and proceeds most rapidly in the cancellous or most vascular parts. This, it is true, is often accompanied by some addition to the exterior in the form of bony outgrowths into the periosteal and tendinous surroundings; but these are slight and by no means compensate for the absorption within and the loss of weight attendant thereon. It is this absorption and thinning of the cancelli, upon the strength as well as the perfection of arrangement of which the upper part of the thigh-bone is much dependent, that renders fracture in that situation so liable to occur in elderly persons. I can only suppose that fatty growth dominates in the skeleton generally more than it does in the skull, and that the same failure of nutritive force which leads in some cases to *bone-formation* in the latter, leads to *bone-absorption* and fatty degeneration in the former.

The other change incidental to age which is also pecu-

liar to the skull is ATROPHY taking place from the *exterior*, whereby the bones are rendered thinner and the cranium proportionately smaller. This is common to all the bones of the skull, affecting the maxillary bones in an especial degree, and the other facial bones more or less, all these becoming reduced in calibre as well as in thickness of their walls, and the face becoming proportionately smaller. In the calvarial parts the change is usually more marked than in the rest of the skull-wall. The outer table recedes, encroaching upon the diploë, and approaching or coalescing with the inner table, so that the bone may be composed of only one thin brittle table. It is a curious process by which this change takes place, for the absorption of the outer table is not attended with any roughening of the exterior. Absorption and deposition go on together, almost at the same spot. While the outer hard laminæ are being removed by the former, the subjacent laminæ are becoming condensed by the latter, and when these again become the subjects of absorption the layers next beneath them become the seat of deposition. Similar changes are observed in the bones of the skull and of other parts when absorption is caused by pressure, as by tumours and sometimes by aneurysms, the lowered or depressed surface being usually smoothed by a filling-in of the cancelli accompanying or preceding the removal of the exterior, and accordingly the part looks as if it had been pressed or beaten in, and so differs from the rough, ragged, gnawed condition caused by cancer or ulceration.

In some instances this absorption takes place uniformly, the several parts of the skull-wall becoming equally thinned, and the entire skull being reduced in size and still more in weight, as shown by the following examples in the Cambridge Museum: An edentulous skull with lower jaw weighs only 15 oz., and the greatest circumference is 19¾ inches. It is very thin, yet the diploë is in fair proportion. There is some recent bone-deposit in the interior, and the meningeal grooves are large. Another, without the lower jaw, and with a circumference of

19½ inches, weighs 11½ oz. A third, from a very aged female, with the lower jaw, weighs 14 oz., the greatest circumference being 20¼ inches. The entire skeleton of this person weighs only 88 oz., though it is evidently that of a fine person, inasmuch as it measures 5 feet 8 inches, the thigh-bones measuring 18¼ inches and the angles of the neck with the shaft being 130°.

What are the causes which determine the incidence of one or the other of these very opposite changes—increase of thickness, with commonly increase of density and weight, on the one hand, and decrease of thickness, with decrease of weight, on the other hand—I cannot tell.

Though commonly, as I have said, the atrophic thinning and removal of the outer table, affects the whole of the calvarial part of the skull in an equal, or nearly equal, manner, yet in some instances it does so very unequally. It has an especial tendency to attack symmetrically the parietal bones between their sagittal or mesial parts and the parietal protuberances, causing those remarkable depressions of which specimens are to be found in most museums, and of which there are nine in the museum at Cambridge and four in the College of Surgeons, one of the latter being a well-formed edentulous Egyptian and one a Wallachian gipsy woman, aged 82, from the Barnard-Davis Collection.[1] They present, on the whole, much similarity, being usually ovoid, measuring three or four inches from before backwards and two or three transversely. At the deepest or middle part the inner layer of the bone may be exposed, reduced to extreme thinness or even quite removed, but it is never, so far as I have seen, indented—that is to say, the inner contour of the skull-wall is not altered. This is shown in Pl. IV, figs. 3 and 4. The surface is smooth, though in a few instances it is slightly rough and marked by vascular foramina; and in the specimen from which Pl. IV, fig. 4, is taken it is traversed by

[1] These were described by Mr. Eve at the Pathological Society ('Lancet,' February 22nd, 1890, p. 404). One of the Cambridge specimens is an edentulous ancient Egyptian skull.

grooves for the meningeal vessels which emerge from the sides and have come to appear on the exterior of the skull. The circumference rises, or shelves, rather suddenly. This is least marked in front and behind, and most marked at the outer border, which often reaches, but does not exceed, the temporal ridge; and the outer border is nearly straight, whereas the inner one is more convex.

These depressions are met with in every stage from a slight, scarcely perceptible, alteration of the normal level to a thinning down to, or through, the inner wall. I am not aware that they are attended with any symptoms or productive of any ill result, though they may render the effects of a blow serious or even fatal. This was shown by a case under the care of Mr. Wherry. A lady, aged 90, fell upon the back of her head, was taken up insensible, and soon died. The parietal depressions, as seen in Pl. IV, fig. 4, are unusually large and extensive, and numerous fractures had taken place through them and into the surrounding bones. They are of irregular shape, and there are islands in which the bone has been less removed than in other parts. In a few instances they are accompanied by similar depressions in other parts of the skull, occasionally in the frontal bone but more commonly in the hinder sagittal parts of the parietals (see Pl. IV, figs. 2 and 3). Some of these latter are more circular, resembling the depressions in the so-called pewter-pot fracture, but without any inflection of the inner table or any fissure; and I have never seen these depressions in other parts so deep as those on the sides of the parietals. In a specimen at Munich the depressions in the usual situation of the parietals are circular in outline; and in one, at Vienna, they are further back than usual, being near the back of the parietals. The depressions look as if the outer layers of the skull had been filed or planed away; but the surfaces are commonly smooth, showing that the process of bone-formation was coincident with that of bone-absorption.

In most instances the skulls thus affected are thin and light, the thinning having taken place from the exterior, so

that the canals for the meningeal vessels are nearer the external surface than is normal; and these canals are often deepened internally by some bony deposit which extends more or less over the whole of the interior of the skull; and, in the specimen represented in Pl. IV, fig. 4, as already noticed, they have, by virtue of the absorption on their exterior and deposition on their interior, come to be on the outer surface of the thin layer which remains at the bottom of the depressions; and they are seen passing on it to the thicker edges at the margins of the depressions, where they disappear. These canals are quite as large and abundant as usual, or more so; there is therefore no evidence of diminution of vascularity at the parts affected or elsewhere.

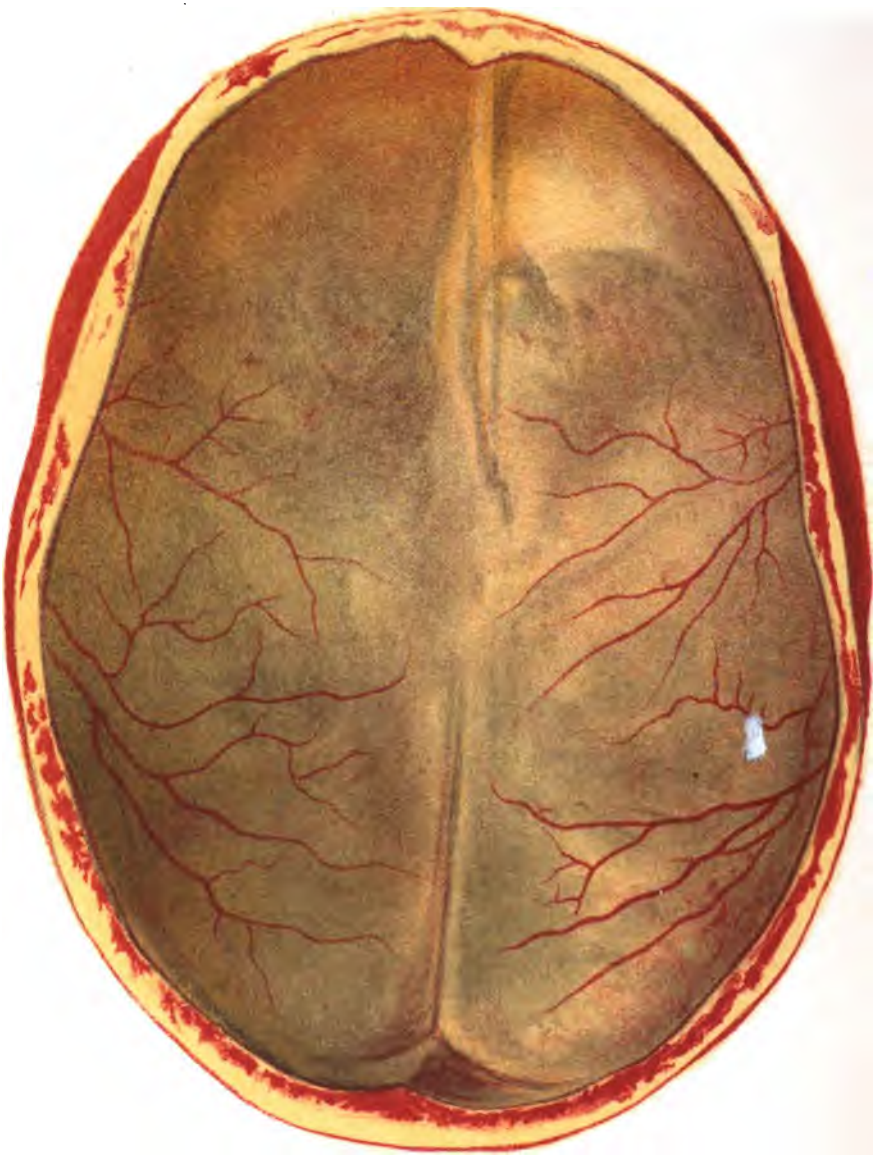
It is further to be observed that the absorption or atrophy which produces these depressions may be associated in the same skull with the opposite, viz., thickening and condensation or hypertrophy. One of the specimens in the Cambridge Museum, a calvaria which I took from a woman, aged 73, who died of apoplexy, is very thick, half an inch in the frontal part, also dense and heavy, weighing 18 oz. The thickening is evidently due to bone-formation on the interior which, especially in the frontal region, is remarkably uneven, knotty, and craggy. The parietal depressions, which occupy the usual position and present the usual features, have not reached the internal table because it has receded from them; and the skull-wall at their deepest part has about the normal thickness and more than the usual density. In the specimen from the woman, aged 90 (Pl. IV, fig. 4), where the depressions are so large, the frontal bone is denser than usual and is thickened with hard knots or tubercles on the interior; and there is similar deposit in the vicinity of the depressions, though the depressions themselves are free from it.

I have been much puzzled to account for these remarkable and symmetrical parietal depressions—these freaks, as it were, of senile process. That they are the result, not, as I once thought possible, of some congenital defect, but

of senile and probably atrophic process, I can no longer doubt, for all the complete skulls in which I have seen them are edentulous, and give other evidence of senile change. That the excesses in the atrophic process are not altogether confined to this particular situation is shown by the occasional occurrence of similar depressions in other parts, more particularly in or near the sagittal suture. But there must be some special cause for this part of the parietal bones being so liable to it and for its advancing here so much more than elsewhere. The cause does not seem to be related to anything in the development, the growth, the texture, the blood-supply or the nutrition of the part; nor to its being subject to the great variation of level, observed by comparing the ill-filled skull of the negro with the well-expanded oval dome of the European and with the squeezed-out parietals in the flattened heads of South American tribes. It cannot be said, as suggested by Mr. Eve, to be the part last ossified or to be in the situation of the parietal foramen. It is indeed the part into which ossification early spreads as it advances from the central parietal protuberance towards the middle line.

In default of other cause, it seems to me that the pressure of the occipito-frontalis tendon, stretched upon and playing over this the most prominent part of the vertex, deserves consideration. The appearance of the depressions is suggestive of pressure; and their shelving front and hinder edges are suggestive of pressure from this source; while their outer margins, which are nearly straight, are limited to the range of the tendon of the occipito-frontalis, and do not ever exceed it. Some countenance is given to this view by the observation in some senile skulls of deep depressions, though I believe these are to some extent inbendings, in the fore part of the temporal fossæ, which are obviously due to the pressure of the thick anterior portions of the temporal muscles.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 122.)



DESCRIPTION OF PLATE III.

Senile Hypertrophy and Senile Atrophy of the Skull (GEORGE MURRAY HUMPHRY, M.D., F.R.S.).

Calvarial part of skull of an alcoholic man, æt. 50, who died of apoplexy, showing congestion of the inner table, which, at parts, was very marked. At all these parts the interior of the skull was thickened by bony deposit causing elevations of the inner lobe.

DESCRIPTION OF PLATE IV.

Senile Hypertrophy and Senile Atrophy of the Skull (GEORGE MURRAY HUMPHRY, M.D., F.R.S.).

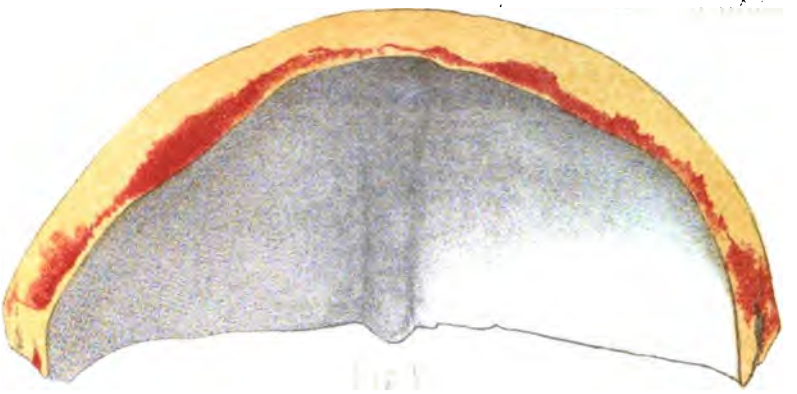
FIG. 1.—Section of Plate III.

FIG. 2.—Skull, showing depressions on parietal bones between sagittal parts and tubera parietalia, also one in middle line.

FIG. 3.—Section of the same through the median and lateral depressions.

FIG. 4.—Effects of extensive absorption taking place somewhat irregularly on parietal bones. The patient, a woman æt. 90, died from fractures through the thinned bones caused by a fall.

The view is from behind, and the fore part is much foreshortened.



A CONTRIBUTION

TO

THE CHEMISTRY OF GOUT.

BY

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The chemistry of gout centres round the properties of uric acid and the urates. It has long been known that gouty concretions are composed of sodium biurate; and in 1848–54 Sir Alfred Garrod demonstrated the important fact that the blood and morbid effusions of gouty persons are abnormally impregnated with uric acid. No substantial addition to our knowledge of the chemistry of gout has since been made.

In a paper recently read before this Society I gave an account of the combinations which uric acid forms with bases.[1] It was there shown that uric acid forms three distinct orders of salts, namely, neutral urates, biurates, and quadrurates. The neutral urates are not known to exist in the body. They can only be produced (artificially) in the presence of the caustic alkalies, free from carbonates, and as such conditions never occur in the living economy,

[1] "On the History of Uric Acid in the Urine," &c., communicated March 25th, 1890. It is assumed throughout the present paper that the reader is acquainted with the second section of my previous paper dealing with the composition and reactions of the quadrurates.

the neutral urates cannot, with our present knowledge, be assumed to take any part in the uratic phenomena of gout.[1] The biurates, although existing pathologically in gouty concretions, are not known with certainty to exist in the healthy or physiological state. The quadrurates, on the other hand, appear to be in a special sense the physiological salts of uric acid. They constitute exclusively the combination in which uric acid exists in normal urine; and, in animals which eliminate their nitrogen as uric acid, like birds and serpents, the urinary excretion is composed entirely of quadrurates. It is, moreover, susceptible of proof that uric acid in liquids containing alkaline carbonates and phosphates, such as are the serum of the blood and its derivatives, enters into solution in the first instance as a quadrurate. From these considerations it may be inferred that in the normal state uric acid is primarily taken up in the body as quadrurate, that it circulates in the blood as quadrurate, and that it is finally voided with the urine as quadrurate; and that when uric acid makes its appearance in any other guise, whether as crystalline biurate in gouty deposits or as free uric acid in gravel, this event is due to secondary and abnormal changes in the quadrurate.

In the paper referred to I traced the changes undergone by the quadrurate in the urine which lead up to the separation of uric acid in the free state as gravel and urinary sediments. In the present paper I propose to trace the converse changes which the quadrurate undergoes in the blood, and which lead up to the formation and deposition of sodium biurate in the tissues of the body. These latter changes are, I think, intimately connected with the property possessed by the quadrurates of slowly taking up, in the presence of the alkaline carbonates and phosphates, an additional atom of base, and of being thereby converted into biurates. A knowledge of this property permits a coherent view to be presented of the succession of events

[1] For this reason the notion of W. Ebstein that the neutral urates are concerned in the formation of gouty deposits appears to be untenable.

which culminate in the gouty paroxysm, and which may be expressed in the following terms.

In the normal state the uric acid, which circulates in the blood as quadrurate, is removed unchanged by the kidneys, and is removed with sufficient speed and completeness to prevent any undue detention or any accumulation of it in the blood. But in the gouty state—either from insufficient kidney action, or from increased introduction of urates into the circulation, or from some altered quality of the blood itself—the quadrurate lingers unduly in the blood and accumulates therein. The detained quadrurate, circulating in a medium which is rich in sodium carbonate, gradually takes up an additional atom of base, and is thereby transformed into sodium biurate. This transformation alters the physiological problem. The uric acid, or rather a portion of it, circulates no longer as the highly soluble and presumably easily secreted quadrurate, but as biurate, which, as we shall see, is almost insoluble in blood-serum, and is, moreover, probably, and for that reason, difficult of removal by the kidneys. Under these new conditions sodium biurate accumulates more and more in the blood, and when the accumulation has reached a certain point it is precipitated in the crystalline form in the joints and elsewhere, thereby determining the occurrence of a "fit of the gout."

The evidence on which this view is based was obtained from a study of the behaviour of uric acid and the urates under various conditions in different media.

It is obvious that the reactions of uric acid and the urates which concern us in gout are not their reactions with simple water, but their reactions with the alkaline and saline media in which they exist and circulate in the body, namely, in the blood and lymph; and herein lies a radical distinction, inasmuch as the reactions in question are wholly different according as the medium is water on the one hand, or blood and lymph on the other.

The serum of the blood and its cognates, the lymph, synovia, and interstitial juices, are closely allied in chemical

composition. Besides albuminoid matters, blood-serum contains certain saline ingredients, on which its behaviour with uric acid and the urates essentially depends. The sodium salts are present in proportion of about 0·7 per cent., the potassium salts in the proportion of about 0·06 per cent., and the calcium and magnesium salts together in the proportion of about 0·05 per cent. It is thus seen that the sodium salts exceed all the other salts put together in the proportion of about seven to one. And practically, for our present purpose, we may consider the saline basis of the blood-serum as consisting essentially of sodium salts, so greatly do these preponderate over the sum of all the other salts put together. The most abundant of the sodium salts is the chloride, which is present in a very constant proportion of 0·5 per cent. The next most abundant salt is the sodium carbonate, probably, chiefly at least, in the condition of bicarbonate. This latter is present in the proportion of about 0·2 per cent. The third sodium salt is the phosphate, which, however, is only present in the proportion of about 0·03 per cent. The serum has always a sharply alkaline reaction, which is due to the large proportion of sodium carbonate contained in it.

From these particulars it may be gathered that a watery solution containing 0·5 per cent. of sodium chloride and 0·2 per cent. of sodium bicarbonate would be a fairly exact imitation of the blood-serum in so far as its saline ingredients are concerned. And it was found experimentally that such a solution behaved in regard to uric acid and the urates in the same manner as blood-serum itself, and in the same manner as a solution composed of all the salines of the serum in their due proportion, as ascertained by the best analyses. A solution was therefore prepared in distilled water, containing 0·5 per cent. of sodium chloride and 0·2 per cent. of sodium bicarbonate. This was called the "standard" solution or solvent. The behaviour of uric acid and the urates with this solution was studied in detail, under varying conditions of temperature and time, and with varying modifications of its composi-

tion. The results thus obtained were then collated with those obtained with blood-serum in similar circumstances, and with parallel modifications of its composition.

By this method of investigation a considerable amount of information was acquired, bearing on the chemistry of the gouty state, and on the genesis of the uratic phenomena of the complaint. The results of the inquiry are considered in the following order. First, the behaviour of the material of gouty concretions, namely, the biurate of sodium, with the standard solvent and with blood-serum is examined. Second, the strongly contrasted behaviour of uric acid and the quadrurates with the same media is followed out. Lastly, some of the conditions which hasten or retard the precipitation of sodium biurate are investigated.

I. Behaviour of Sodium Biurate with the Standard Solvent and its Modifications, and with Blood-serum.

When sodium biurate[1] is digested, at blood heat, with pure water it enters pretty freely into solution. Such a solution acidulated with hydrochloric acid throws down a copious precipitate of uric acid. Careful experiments indicated that the solubility of sodium biurate in distilled water, at 100° F., fully amounts to 1 part in 1100. But when sodium biurate was digested at the same temperature with the standard solvent very little went into solution—so little that 100 c.c. of the filtered product, after treatment with hydrochloric acid, only yielded a few scattered crystals of uric acid—a quantity too small to be weighed.

[1] Sodium biurate was prepared by boiling 4 grams of uric acid in 400 cubic centimetres of a 1 per cent. solution of sodium bicarbonate. This was filtered hot and then allowed to stand for twenty-four hours. A copious precipitate of crystalline stars and needles was thus obtained. This was thrown on a filter and washed with cold distilled water, and then dried at 100° F. In experimenting with the urates the investigator should always prepare his own materials. Specimens of the urates supplied to me by dealers proved to be mere crude mixtures of uric acid with the bases, and were wholly unfit for exact experiments.

I estimated that the solubility of sodium biurate in the standard solvent at 100° F. could certainly not exceed 1 part in 10,000. It was, moreover, found that no addition to the solvent of any salts—whether of potassium, lithium, or magnesium—whether as carbonates, chlorides, phosphates, salicylates, iodides, or bromides—made any appreciable difference.[1] On the other hand, if the standard solution was modified in the opposite direction—in the direction of subtraction—its solvent power progressively increased. In other words, the nearer the solution approached to pure water the higher became its power of dissolving sodium biurate; and, on the contrary, the richer it was in sodium salts the more was its solvent capacity reduced.

The solvent power of the solution seemed to be determined exclusively by the sum of sodium salts contained therein; it mattered little, so long as the quantity of base was constant, what the combination was. The degree of alkalescence had not the slightest influence, and a solution of sodium chloride or sulphate was absolutely on a par with a solution of sodium carbonate containing the same amount of base.

Solutions of the salts of potassium, lithium, and magnesium, containing from 0·1 to 0·5 per cent., dissolved sodium biurate about as freely as distilled water, and consequently very much more freely than equivalent solutions of the sodium salts. These advantages as solvents were, however, completely nullified when the potassium, lithium, and magnesium salts were used not alone, but as additions to the standard menstruum—that is to say, in presence of 0·7 per cent. of sodium salts. Comparison of the chloride with the carbonate of potassium showed that the carbonate had not the least advantage over the chloride.

Salts of calcium and ammonium were found to act, in regard to the point under consideration, in the same way and in about the same degree as salts of sodium—that is

[1] The additions made varied from 0·1 to 0·5 per cent.

to say, they lessened the solvent power of water on sodium biurate in proportion to the strength of their solutions.

The behaviour of sodium biurate with the serum of the blood was next examined. In blood-serum sodium biurate was found to be even less soluble than in the standard solution, as the following observations indicate.

Experiment 1.—Sodium biurate in excess was digested with serum of pig's blood, at 100° F., for twenty-four hours, with frequent agitation. The serum was then twice filtered through a threefold filter.[1] Of this product 50 c.c. were acidulated with strong acetic acid and set aside. After standing forty-eight hours no crystals of uric acid were found to be deposited. A second portion was carefully tested by Sir Alfred Garrod's uric acid thread experiment. Only a few crystals were found sprinkled on the thread. A third portion was simply evaporated, at 100° F., to the consistence of a thick syrup. In this needles of biurate were easily detected under the microscope. These observations showed that sodium biurate, although very sparingly soluble, is not absolutely insoluble in healthy serum.

Experiment 2.—Three metatarsal bones[2] from the body of a gouty man, which were encrusted on their articulating surfaces with uratic deposits, were treated as follows:—The first (A) was suspended in six ounces of distilled water, the second (B) was suspended in a similar quantity of the standard solution, and the third (C) in the same volume of blood-serum. The phials in which the specimens were contained were tightly corked, and their contents preserved from putrefactive changes by the inclusion of thirty or forty drops of chloroform. They were placed in the warm chamber for a fortnight, and after that were kept at the temperature of the room. In four days the

[1] The crystalline needles of sodium biurate are so minute and delicate that a portion passes through the filter unless extraordinary precautions are taken.

[2] Kindly presented to me by Dr. Norman Moore. The deposits were tested and found to be composed of urates.

deposit was entirely dissolved out from the first specimen (A). The second specimen (B) showed distinct signs of solution in fourteen days, and at the end of six weeks the deposit had entirely vanished. The third specimen (C), suspended in serum, still remains, at the end of eight months, apparently unaltered; the limits of the encrusted spots are as sharply defined, and the quantity of the deposit appears as great, as when the preparation was first put up.

It was found that the addition to blood-serum of small quantities (0·1 to 0·5 per cent.) of the carbonates, chlorides, or phosphates of potassium, sodium, or lithium did not in the least degree enhance the solvent power of the serum on sodium biurate.

II. Behaviour of Uric Acid with the Standard Solvent, and with Blood-serum and Synovia.

Observations on the Standard Solvent and Blood-serum.

The reactions of uric acid with the standard solvent and with blood-serum stand in the strongest contrast with those of sodium biurate in the same media.

When uric acid is digested with the standard solvent or with blood-serum it passes freely into solution in combination with a base. The compound thus formed is, in the first instance, undoubtedly a quadrurate.[1] But the process does not stop here. The quadrurate gradually takes up an additional quantity of base, and is thereby converted into biurate, and the biurate thus formed is, after some delay, eventually precipitated in the crystalline state. In studying this process it is desirable to distinguish two, if not three stages, namely, first, the taking up the uric acid as quadrurate, or *solution*; second, the transformation of quadrurate into biurate, or *maturation*. This

[1] The experimental evidence on which this conclusion is based is given in my previous paper published in the present volume.

latter stage culminates in the precipitation and deposition of the biurate in the cystalline form, and this should perhaps be considered as a third stage, or *precipitation*.

The following experiments may be taken as illustrations of the method of experimentation pursued and of the results obtained.

Experiment 1.—A gram of uric acid was introduced into a flask with 200 c.c. of the standard solvent. The flask was tightly corked and placed in the warm chamber, where the temperature was continuously maintained at 100° F. A considerable amount of uric acid went into solution, but a portion remained undissolved at the bottom of the flask, leaving a clear supernatant liquor. Things remained apparently unchanged until the evening of the second day, when a few stars of biurate were detected amid the undissolved sediment of uric acid. On the third day, however, a rapid change was observed to be taking place, consisting in an abundant precipitation of stars and tufts and detached needles of biurate. On the fourth day the precipitation appeared to be nearly complete, for the supernatant liquor now showed only small traces of uric acid when it was treated with hydrochloric acid.

Experiment 2.—A parallel experiment was made with blood-serum. Fresh serum of pig's blood was treated with uric acid in excess in a 4-oz. phial, tightly corked and chloroformed to prevent decomposition. The phial was gently turned upside down a few times at first, and was not subsequently disturbed. It was then placed in the warm chamber at 100° F. The serum soon cleared, the surplus uric acid fell to the bottom, and the supernatant serum became transparent. For about twenty-four hours no change occurred, but in the course of the second day stars of biurate were detected amid the deposit, and during the third day an abundant precipitation of stars, tufts, and needles of biurate took place, exactly resembling those found in gouty concretions. On the fourth day the process of precipitation was nearly complete, and

the supernatant serum was found to be comparatively free from uric acid.

In order to isolate the stage of maturation from that of solution the experiments were modified in the following manner:

Experiment 3.—Uric acid in excess was digested with frequent agitation with the standard solvent, at 100° F., for twenty minutes. The excess of uric acid was then filtered off, and the clear solution was placed in a corked phial in the warm chamber. It remained unaltered for two days. On the third day it began to precipitate, and on the fourth day a copious deposition of crystalline biurate took place. On the fifth day the process was completed, and the supernatant liquor was found on acidulation to contain only traces of uric acid.

Experiment 4.—Blood-serum of the horse was digested, at 100° F., with excess of uric acid for fifteen minutes with constant agitation. It was then filtered and placed in a corked phial in the warm chamber. In about twelve hours the serum, previously clear, began to lose transparency, and fine needles of biurate were detected in it with the microscope. On the next day copious precipitation took place. On the fourth day the process seemed to be completed, and the supernatant serum was found to be comparatively free from uric acid.

It was impossible not to be struck with a certain rough resemblance between the results observed in these experiments and the phenomena of the gouty paroxysm. In the gouty subject it is assumed that the blood becomes more and more impregnated with uric acid until, after a certain period of incubation has been accomplished, sudden precipitation of sodium biurate takes place in and about the joints, and the "fit of the gout" is declared. Then follows a process of recovery, with restoration of the blood to a purer state—that is, with a lessened impregnation with uric acid. In the artificial counterfeit we observe a similar succession of events: firstly, impregnation of the medium with sodium quadrurate; secondly, a period of in-

cubation or maturation, during which the quadrurate passes into biurate; thirdly, somewhat sudden precipitation of sodium biurate in the crystalline form; and lastly, restoration of the medium to comparative purity.

In the above-recorded experiments the quantity of uric acid in solution was not accurately gauged, but in the light of subsequent experiments I judge it to have been about 1 part in 1500. The speed of maturation (the length of time intervening between solution and precipitation) was found to be greatly influenced by the percentage of uric acid in solution. The richer the medium was in uric acid, the more quickly was maturation consummated, and the earlier was the occurrence of precipitation. In the case of blood-serum, when the impregnation with uric acid amounted to 1 in 800 or 1 in 1000, the first beginnings of precipitation were observed in four to six hours, and copious critical precipitation took place in twelve to fourteen hours. On the other hand, when the proportion of uric acid in solution was only 1 in 4000 or 1 in 5000, precipitation was delayed for six to twelve days, and was even then so slight in amount as to be only detectable by microscopic examination. It was further noted, in these feebler solutions, that the deposition of crystals was throughout gradual, and that there did not occur any sudden or critical fall of crystals, such as was observed to take place in the middle periods of maturation in the stronger solutions. Some of these points will be again adverted to in treating of the circumstances which hasten or retard maturation.

Observations on Synovia.

It is a marked peculiarity of the uratic phenomena of gout that the deposits occur most commonly in and about the joints; and it was a matter of interest in the present inquiry to ascertain how the synovial fluids which bathe these parts behave when impregnated with uric acid, and especially whether these fluids, as compared with blood-

serum, and when impregnated to an equal degree, had the property of promoting or hastening the precipitation of biurate. This part of the investigation is beset with difficulties, and my information thereupon is very scanty. The synovial pouches are shut sacs, entirely detached from one another, and it is conceivable that slight differences in the composition and the percentage of their saline constituents may exist between them, and that such differences may account for the preferential order in which the joints are attacked in the gouty paroxysm. The quantity of synovia obtainable from the smaller joints is very scanty, even in the case of the large quadrupeds which are dealt with in our slaughter-houses. I have so far only been able to make satisfactory experiments with synovial fluid drawn from the hip of the ox. From this source about a couple of ounces may be obtained from the two joints. Butchers tell me that the synovia from the hip is thinner as well as more abundant, than that obtained from the other joints.

On two occasions I was able to examine and compare the synovial fluid of the hip-joint with the blood-serum of the same ox. The behaviour of the two fluids with uric acid was substantially the same, but in both instances I found that, when impregnated with uric acid in an equal degree, precipitation of biurate began distinctly a little earlier in the synovia than in the serum. And my impression is, but it is only an impression, that with the thicker and more concentrated synovia of the smaller joints this difference would be more pronounced. Whether there is in this distinction between serum and synovia a key to the preference of uratic deposition for the joints is a question well worthy of further inquiry. It is at any rate conceivable, supposing the blood and its derivative fluids to be equably impregnated with uric acid, and supposing the synovial fluids to be more largely charged than their congeners with salts, and especially with sodium and calcium salts, that precipitation of biurate would take place earlier, and by preference, in and about the joints than elsewhere.

Behaviour of the Quadrurates with Blood-serum.

The quadrurates behave with the standard solvent and with blood-serum substantially in the same way as uric acid. This might have been expected from the fact that these media take up uric acid in the first instance as a quadrurate. There is, however, this difference, that the quadrurates pass into solution much more rapidly than uric acid, and, consequently, the period of precipitation of biurate has its advent considerably accelerated.

III. The Conditions which Accelerate or Retard the Processes which culminate in the Precipitation of Sodium Biurate.

Assuming a real analogy to exist between the processes which go on in serum artificially impregnated with uric acid, and the processes which go on in the blood of a gouty patient, and which culminate in the deposition of uratic concretions, it is a matter of interest, as bearing on the pathology and treatment of gout, to investigate the conditions which, in the artificial parallel, accelerate or retard these processes. In pursuing this study from our chemical standpoint the three stages or phases in the development of the gouty paroxysm should be kept distinct in the mind, namely, the stages of solution, maturation, and precipitation. The juvantia and obstantia of these three stages are necessarily different and require separate consideration.

The gouty man may be regarded as living on the brink of a critical outbreak. His uric acid function is in a state of unstable equilibrium, and it is conceivable that a little quickening or favouring circumstance in any of the three stages might determine the occurrence of a paroxysm which would not otherwise have taken place. In the gouty state it is probable, as Dr. Haig suggests, that there may be stores of uric acid (or quadrurates) lodged in certain organs, as the spleen or liver, or diffused more generally through the tissues of the body. An elevation

of the dissolving power of the lymph or blood, acting on these stores, might lead to a sudden irruption of urates into the blood, transcending the capacity (perhaps already impaired) of the kidneys for their elimination, and so lead to an outbreak. In like manner some new conditions in the blood, some variation in its chemical or physical properties, might hasten or retard the other stages of maturation and precipitation, and thereby determine the occurrence or non-occurrence of a gouty paroxysm.

The rising and falling activity of the kidneys, in the matter of separating the urates floating in the blood, does not come within the scope of the present inquiry, though doubtless a potent, if not the most potent, factor in the genesis of gouty explosions and of gouty phenomena generally.

Conditions affecting the Stage of Solution.

It may be stated generally that the more alkaline the medium is, the more rapidly and freely does it dissolve uric acid and quadrurates. This is certainly the case with solutions of the alkaline carbonates and phosphates, ranging from 0·5 to 5 per cent. The neutral carbonate is a better solvent of uric acid than the bicarbonate. It is believed that sodium carbonate circulates in the blood partly as neutral and partly as acid carbonate, and that the proportion varies. If this be so a rising proportion of neutral carbonate would promote, and a rising proportion of bicarbonate would retard, the solution of uric acid stored in the tissues.

The solvent power of the blood on uric acid depends exclusively on the alkaline carbonates and phosphates contained in it. The neutral salts—chlorides and sulphates—were not found to have the least influence either way on the act of solution.

The quadrurates are taken up more rapidly by blood-serum than free uric acid. The following experiment illustrates this point. Half a gram of uric acid and the same quantity of serpent's urine (which is composed of

almost pure quadrurates), were separately digested in 200 c.c. of blood-serum, without agitation, at 100° F. In the latter case all went soon into solution, and abundant precipitation of biurate took place in twenty-four hours. In the former case solution was much slower; and precipitation did not begin until the third day, and was not abundant until the fourth day.

Conditions affecting the Stages of Maturation and Precipitation.

The investigation embraced a study of the effects of temperature, percentage of uric acid in solution, and the addition of various saline substances to the maturating medium.

(A) *Temperature.*—It was found invariably that the stage of maturation was more quickly accomplished in the warm chamber at 100° F. than at the temperature of the room, ranging from 60° to 70° F.; but the ultimate result was exactly the same in both cases. For example, serum charged with 1 part of uric acid in 600 began to precipitate in the warm chamber in four hours, and precipitated copiously in six hours. A duplicate specimen kept at the temperature of the room (65° F.) began to precipitate in eight hours, and did not precipitate copiously for sixteen hours. Another sample of serum, impregnated with 1 part of uric acid in 1000, began to precipitate in the warm chamber in six hours, and deposited copiously in fourteen hours; while a duplicate kept at the temperature of the room (60° to 70° F.) only began to precipitate in thirty hours, and copious precipitation did not take place for forty-eight hours.

The absolute constancy of these results led to the idea that maturation would go on more rapidly at a febrile temperature (104° to 105° F.) than at the normal temperature of the body, and that herein might be found an explanation of the circumstance that gouty outbreaks

sometimes follow immediately on the heels of an injury. When, however, this notion was tested experimentally no support was found for it.

It was also conceived that although maturation itself was favoured by warmth, the terminal act of the process, namely, the act of precipitation, might, on the contrary (seeing that sodium biurate is more soluble at higher than at lower temperatures), be favoured by cold, and that this might account for the fact that gouty concretions tend to be deposited in the cooler and more exposed parts of the body, in the joints and subcutaneous tissues, rather than in the warmer interior regions. I failed, however, to obtain any direct experimental evidence in favour of this conception.

(B) *Quantity of uric acid in solution.*—It was found that no condition exercised so great and decisive an influence on the speed of maturation and the advent of precipitation as the proportion of uric acid in solution. The amount or copiousness of the precipitation was likewise, of course, affected in the same way. The following experiment with blood-serum, the results of which are arranged in a tabular form, illustrates these points in a striking manner. The phials containing the serum were placed in the warm chamber for fourteen days, and were afterwards kept at the temperature of the room. Chloroform was added to prevent putrefactive changes.

Table showing the influence of percentage of uric acid in solution on the speed of maturation, and the time of advent of precipitation.

Quantity of uric acid contained in the serum.	Time of precipitation of sodium biurate.
1 in 1000 . .	Precipitation began in 6 hours. Copious precipitation in 14 hours.
1 in 2000 . .	Precipitation began in 33 hours. Copious precipitation in 3 days.
1 in 3000 . .	Slight precipitation began in 3 days, which became a little more copious in 12 days.

Quantity of uric acid contained in the serum.	Time of precipitation of sodium biurate.
1 in 4000 .	. A few needles of biurate were detected on the 6th day; more needles and a few tufts in 12 days.
1 in 5000 .	. A few short needles were detected on the 13th day. In 30 days the needles were somewhat more numerous.
1 in 6000 .	. No needles were discoverable in 14 days; a few were detected in 40 days.
1 in 8000 .	. No needles could be detected after the lapse of 40 days.

Assuming that the inflammatory arthritic attacks in gout are directly due to copious and sudden precipitation of crystalline stars and needles of sodic biurate in the cartilages and fibrous structures of the joints, the evidence before me indicates that such copious sudden precipitation can only take place when the fluids bathing these structures are impregnated with uric acid in, at least, the proportion of 1 part in 2500. Below this point the precipitation occurs slowly and scantily, and only in the form of short scattered needles. When the proportion of uric acid in the serum was only 1 part in 5000 the deposited needles were mostly about as long as the diameter of a red blood-disc, some were twice this length, and a few three times this length, and all were of extreme tenuity. It is quite conceivable that this slighter precipitation in the tissues, of short scattered needles, might account for certain irritations in the various organs, such as characterise irregular or larval gout; but it could scarcely engender downright inflammatory attacks. It is further conceivable that the presence in the blood of such scattered needles might constitute foci, around which clotting might take place; and that the thrombosis not unfrequently observed in gouty cases might thus be accounted for.

The impregnation of the blood in gouty persons with uric acid to the extent of these lesser degrees is within the range of observed actualities. Sir Alfred Garrod obtained, by quantitative analysis, from the blood-serum of

one of his patients uric acid to the amount of 1 part in 5714; and he remarks that the quantities thus recoverable from the blood are probably much under the actual amounts, as considerable loss is liable to occur from unavoidable causes.

These considerations lead to the suggestion that a microscopical examination of the blood in gouty persons might sometimes reveal the existence of needles of biurate in that fluid. I tested this point in ten cases of chronic gout, by examining a drop of blood drawn from the finger, but I failed to obtain positive results.

Addition of Various Saline Substances.

Our ideas on the therapeutics of gout are largely coloured by chemical considerations. It is supposed that the efficacy of mineral springs, which are so much resorted to by gouty patients, depends on the saline ingredients which they contain. The results of the present inquiry throw great doubt on the validity of this notion. The springs in repute, and in equal repute, are of the most varied character. Some are charged with bicarbonate of sodium, others with chloride and sulphate of sodium, others again with salts of lime and magnesia, and a few contain traces of lithia. These salts are supposed, when introduced into the circulation, to have the property of rendering the uric acid floating in the blood more soluble, and of thus promoting its elimination by the kidneys. Under the influence of the same ideas the alkaline carbonates are largely prescribed to gouty patients. It has already been shown that these views, so far as they concern the solubility of the material of gouty concretions, are based on an erroneous assumption. The addition to serum of any of these salts did not in the least degree enhance its solvent power over sodium biurate; and with regard to the salts of soda and lime, their effect was, in this respect, distinctly adverse.

A very considerable series of experiments were made with the purpose of ascertaining whether the addition of saline substances to serum impregnated with uric acid exercised any influence on the progress of maturation and the advent of precipitation. The additions made varied from 0·1 to 0·4 per cent., and the experiments were carried out in the warm chamber at 100° F. The salts tried were the carbonate, chloride, sulphate, phosphate, and salicylate of sodium; the carbonate, chloride, and phosphate of potassium; the carbonate of lithium; the chloride and sulphate of magnesium; and the chloride of calcium. The salts of soda and lime were found to accelerate the process, and to hasten the advent of precipitation. The carbonate and phosphate of potassium, the carbonate of lithium, and the sulphate and chloride of magnesium were indifferent. The addition of 0·1 per cent. of the chloride of potassium appeared to sensibly postpone the advent of precipitation. I obtained no evidence in support of the assumption that increasing the alkalescence of the blood lessened the tendency to the deposition of uratic concretions.

The impression produced by the inquiry in regard to the use of mineral waters in gout was to the effect that their virtues depended a good deal more on the water they contained than on their saline constitutents, and that the springs which were most likely to be of service were those which approached nearest to pure water.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 132.)

ON

FOUR HUNDRED CASES OF AMPUTATION

PERFORMED AT ST. GEORGE'S HOSPITAL, FROM OCTOBER, 1874, TO JUNE, 1888;

WITH ESPECIAL REFERENCE TO THE DIMINISHED RATE OF MORTALITY.

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To vol. i of the 'St. George's Hospital Reports,' published in 1866, Mr. Holmes contributed a paper dealing with 300 cases of amputation. The statistics were obtained from the "Amputation Book," in which are recorded particulars of all amputations performed since the year 1852. Partial amputations of the hand and foot, which are usually performed by the house surgeons, are not included, but complete operations, such as Lisfranc's or Chopart's, find a place. Vol. viii of the same 'Reports,' published in 1878, contained a second paper also by Mr. Holmes on a further series of cases, 200 in number. In this second paper Mr. Holmes made some remarks on the antiseptic treatment of cases of amputation.

We propose now to consider a third series of 400 cases, dealing with the amputations performed between October, 1874, and June, 1888, following the lines that were laid down by Mr. Holmes in order to facilitate comparison of results. We have endeavoured to set forth the statistics in the plainest possible manner, and to let the figures, as far as may be, speak for themselves. The record is a continuous one. The three series, in fact, comprise a total of 900 consecutive amputations performed by twelve surgeons at one hospital during a period of thirty-six years. It follows, therefore, that to a limited extent only can the figures be taken to show the difference between the results obtained before and those subsequent to the general adoption of the antiseptic system. Our cases date from October, 1874. Previously to 1880 the records only occasionally note the employment of antiseptic treatment on the Listerian method. In 1876, for instance, silken ligatures were very commonly employed. Usually one end was cut off short, and the long ends, gathered together in a bundle, were brought out at the corner of the wound, providing in this way to some extent for drainage, though the importance of this principle was not perhaps so fully recognised as it is now. Layers of lint soaked in carbolic acid solution were usually laid over the wound and covered by oiled silk. At the conclusion of an amputation the wound was frequently syringed out with cold water or with iced carbolic solution. This latter detail was by no means the invariable practice, but it was certainly in 1876 one very frequently employed as far as amputations were concerned. Due attention was paid to cleanliness, but no attempt was made, by soaking in antiseptic fluids, to render the instruments or ligatures or the hands of the surgeon aseptic. Cases of other kinds were often treated on the Listerian method. The system had indeed been employed, as in other hospitals, for some years.[1] In 1877 catgut ligatures came into general use.

During the period 1877–9 the methods were constantly

[1] 'St. George's Hospital Reports,' vol. iii (1868), p. 241.

changing. Taking a few cases almost at random from our records for 1878 and 1879, we find, for example, the following notes on the methods and dressings employed: —"Antiseptic method throughout; catgut ligatures; drainage." The next :—"Carbolic dressings; drainage-tube; silk ligatures." In some cases dry dressings were used, and in a very few irrigation of the open wound was practised.

In 1880 minute attention was paid to all detail which might be considered to affect injuriously the asepticity of an amputation wound. At this period and for some years subsequently the carbolic spray was generally used for amputations. Of late years it has almost entirely been abandoned, with great advantage. It may be noted here, parenthetically, that since the adoption of catgut ligatures the occurrence of secondary hæmorrhage has been very rare. Two such cases are recorded under Table VI, D. In one of these gangrene had followed fracture of the surgical neck of the humerus in a man aged 71, and the arm was amputated. Secondary hæmorrhage took place on the sixth day, and the patient died on the seventh day. Post mortem the vessels were found to be atheromatous. The other occurred in a patient whose leg was amputated through the knee-joint for a malignant tumour of the leg.

Consecutive hæmorrhage happened once in a way when some vessel that did not bleed at the time of operation was overlooked, but the formidable hæmorrhage due to the ulceration of a large vessel seems to have been almost entirely done away with. Yet this used occasionally to occur with the silken ligatures. When the vessels are diseased and atheromatous, catgut or tendon seems beyond question the safest material at present available.

These 400 amputations, then, comprise a large proportion of cases treated strictly (according to the present state of our knowledge) on the antiseptic system, but also a considerable number in which other, or what would now be called imperfectly antiseptic methods were employed. It is often supposed that the practice of a general hospital

is greatly bound by traditions. To imagine this to be the case is to presuppose a very limited capacity for improvement. The methods in vogue are in reality in a constant state of modification with the view of further improvement. That which might be accepted as nearly perfect to-day may be, almost surely will be, considered worse than antiquated a few years hence. The practice as regards amputations at St. George's Hospital is the same as obtains in all general hospitals. The patient is considered before the amputation book. The possibility —however remote—of saving life or relieving suffering, even if only for a short time, is held paramount. The statistician may employ the figures and tabulate the results, but he has absolutely no influence on the practice.

It is worthy of remark that more amputations were performed during the second than during the first half of the period covered by these statistics. For the six years 1876 to 1881 (inclusive) 146 cases are tabulated, and for the six years from 1882 to 1887 (inclusive), 203. Taking into account the increasing reluctance of surgeons to submit their patients to amputation it is at least probable that these 203 included an even larger number of unpromising cases than the 146. On the whole, however, it is better to assume that the class of cases did not greatly differ from those previously tabulated, though if any advantage could be shown to exist it would probably be found in the earlier series. The hospital was re-floored in 1886-7, hard wood (teak) being substituted for the deal that had stood the wear and tear and scrubbing of many years, but otherwise no substantial alteration was made in the building, and the same number of beds was available. The conditions then under which the present series of amputations was performed were much the same as in former years. Save for the introduction and elaboration of the antiseptic method no potent factor can be held accountable for any alteration in the rate of mortality. That rate, Mr. Holmes concluded,[1]

[1] Vol. i, p. 320.

varies, *cæteris paribus*, with the prevalence of pyæmia. Our investigation clearly proves the truth of this remark, as will be seen later on.

The two main points to which Mr. Holmes directed attention in his first paper were[1]—

1. The influence of advancing age on the results of amputation.

2. The proportion of cases dying from the effects of previous injury and disease to those dying from the sequelæ of the operation.

With regard to the first of these subjects of inquiry we need say little. The following tables furnish at a glance an interesting comparison.

TABLE I.

	First series. Feb., 1859, to Feb., 1866.		Second series. June, 1863, to Oct., 1874.		Third series. Oct., 1874, to Oct., 1888.		Total.		Deaths per cent.			
									1st ser.	2nd ser.	3rd ser.	Total, 3 ser., 900 amp.
	No.	Died.	No.	Died.	No.	Died.	No.	Died.				
Under 5 years . .	1	0	4	1	16	2	21	3	—	25·0	12·5	14·3
Above 5 and under 10	14	1	6	1	30	1	50	3	7·1	16·6	3·3	6·0
„ 10 „ 15	21	1	16	3	28	4	65	8	4·7	18·7	14·2	12·3
„ 15 „ 20	47	8	21	7	44	8	112	23	17·0	33·3	18·1	20·5
„ 20 „ 30	74	14	46	16	95	14	215	44	18·9	34·7	14·7	20·4
„ 30 „ 40	53	21	42	17	63	9	158	47	39·6	40·4	14·2	29·7
„ 40 „ 50	41	15	34	14	58	12	133	41	36·5	41·1	20·7	30·8
„ 50 „ 60	34	17	17	9	40	18	91	44	50·0	52·9	45·0	48·3
„ 60 „ 70	13	5	10	6	20	11	43	22	38·4	60·0	55·0	51·1
„ 70 . . .	2	1	4	1	4	3	10	5	50·0	25·0	75·0	5·0
Adults of unknown age	—	—	—	—	2	2	2	2	—	—	—	100·0
	300	83	200	75	400	84	900	242	27·6	37·5	21·0	26·8

The most noticeable contrast will be observed in the third series of amputations, between the ages of twenty and fifty. The mortality between the ages of twenty and thirty, which in the first series amounted to 18·9 per cent., and in the second series to 34·7 per cent., in the third has fallen to 14·7 per cent. Between the ages of thirty and forty the

[1] Ibid., p. 292.

figures are still more striking, for the mortality as shown in Table I, which in the first series was 39·6 per cent., and in the second 40·4 per cent., has in the third series fallen to 14·2 per cent. Between the ages of forty and fifty much the same results will be observed. The improvement shown in the third series is chiefly due to the diminished mortality in these three divisions.

Of the gross total (900) of amputations tabulated, 506 were performed on persons between twenty and fifty years of age. The third series illustrates in a very emphatic manner the influence of age, for with a greatly diminished gross rate of mortality this influence is as convincingly demonstrated as in the former tables.

The youngest case tabulated was that of a child aged 1½, whose thigh was amputated for gangrene following a simple fracture of the femur. This patient died of pyæmia (Table VI, A, No. 5). The condition may have been due to fat embolism, but there was a wound also of the other foot. The oldest was a woman aged 77, whose forearm was amputated for epithelioma attacking a burn of the hand of sixty-eight years' standing. This patient made an uninterrupted and quick recovery.

The following table (II) shows the ages of the patients in our series of cases :

TABLE II.

	No. of cases.	Deaths.	Per cent.
Under 5 years	16	2	12·5
Above 5 and under 10 . . .	30	1	3·3
„ 10 „ 15 . . .	28	4	14·2
„ 15 „ 20 . . .	44	8	18·1
„ 20 „ 30 . . .	95	14	14·7
„ 30 „ 40 . . .	63	9	14·2
„ 40 „ 50 . . .	58	12	20·7
„ 50 „ 60 . . .	40	18	45·0
„ 60 „ 70 . . .	20	11	55·0
„ 70	4	3	75·0
Adults of unknown age . . .	2	2	100·0
	400	84	21·0

Assuming, for convenience' sake, that of the two adults mentioned in the last line one was under and one over thirty, we have, out of 463 amputations under the age of thirty, 81 deaths; and out of 437 amputations over the age of thirty, 161 deaths, or almost exactly double. This is precisely the result at which Mr. Holmes arrived.[1] The diminished rate of mortality is a general diminution. We shall endeavour to show subsequently that it is chiefly due to the rare occurrence in recent years of pyæmia after amputations, and shall ascribe this to the more perfect employment of the antiseptic system. Nevertheless the influence of age remains the same, and this fact should be noted in questions of prognosis. Three hundred and thirty-nine of the cases in the third series were under fifty years of age, and of these 50 died. Sixty-one were over fifty, and of these 34 died. The contrast is astonishing. Yet these older people had every advantage and security which the most modern and improved methods could give. For all that they died. Whatever the methods employed amputation is about four times as dangerous after the age of fifty as it is before. No doubt the disorders for which amputation is necessary in later life are, on the whole, much graver than in those less than fifty years of age. Chronic diseases of the joints, if the operation be not too long deferred, are eminently favorable for amputation. Such cases are rarely met with in old people, while they constitute the great bulk of the "Pathological" amputations in the young. Gangrene, again, and the like grave disorders swell the mortality in the old. Loss of blood is less well withstood, and the general recuperative power is feebler. A much larger percentage in the old really die from the effects of previous disease than from the operation or its sequelæ. This point will, however, be dealt with later on. It suffices now to note that the concentration of attention on the condition of the amputation wound is not in itself likely to lead to a diminution of mortality in the old after amputation. The older

[1] 'St. George's Hospital Reports,' vol. viii, p. 283.

the patient, the more must he be considered apart from his wound.

The second point to which we desire to draw attention is the proportion of cases dying from the effects of previous injury and disease to those dying from the sequelæ of the operation.

It is, of course, obvious that no improved system of wound treatment is likely to affect this proportion to any very great extent. Indeed, if any influence at all could be expressed, it is possible that it might not be in the direction of improvement as regards the mere figures. A method which aims at and almost invariably succeeds in obviating traumatic fever may lead the surgeon to amputate in extremely unfavorable cases, as, for example, in advanced phthisis. On this second point there is no satisfactory way of illustrating by figures any contrast between our series and those previously tabulated. Yet it can hardly be doubted that some such improvement exists and affects the general diminution of mortality. Improved after-treatment, greater efficiency and skill in nursing, increased watchfulness of detail, must have much to do with the better results, for after all the antiseptic system is directed to the operation wound primarily, and not so much to the patient who has been operated on. The antiseptic system is a most powerful weapon of defence, but it is not a whole armament, and if the figures work out to much the same totals the explanation is probably to be found in the greater severity of the cases operated on. Some support is lent to such an inference by the increased number of amputations shown by our tables to have been performed during the last few years. An appreciable number of these cases would possibly have been considered too hopeless for operation in former years. For instance, the presence of albumen in the urine was at one time held almost sufficient of itself to contra-indicate operation, even amputation. Yet there are many cases in our series, especially those of patients suffering from inveterate bone disease or joint affections of long standing,

who at the time of operation had marked albuminuria, which after operation either entirely disappeared or gradually diminished to a mere trace. Slowly progressing sclerosis of bone attended by much pain is particularly associated with this albuminuria, and the presence of the symptom frequently indicates the necessity for rather than vetoes the amputation.

The results of the various amputations performed may now be analysed to some extent. Attention may again be drawn to the rule which has guided us throughout in estimating the mortality. If a patient who has been the subject of an amputation dies while still on the books of St. George's Hospital the case is reckoned as one of death after amputation, whatever the proximate cause may have been. Clearly, as will be seen by the remarks made below, many cases tabulated as deaths after amputation ought strictly never to have been so entered. Yet it seemed better not to depart in any degree from the plan hitherto adopted, nor to seek to arrange the results so as to give the most favorable impression. A certain number of patients who are entered as "recovered" may have died shortly after leaving the hospital, and thus form a set-off. As an example, a patient with, say, a diseased knee and pulmonary tuberculosis undergoes amputation of the thigh. The stump heals, and the patient dies of phthisis some weeks or months later than would have been the case if he had been dragged down by the pain of the diseased joint. Yet such a case would be, and is in our tables reckoned as, a death after amputation. Again, a similar patient has his arm removed for diseased elbow. The flaps break down, the bone protrudes, the pulmonary trouble if anything is made worse. The patient leaves the hospital wishing "to die at home," and probably does so shortly. The case is entered as a "recovery." Truth to tell, the mortality of a given hospital forms but the coarsest guide to the success of its practice, but no other test can well be applied.

In order to furnish, as far as possible, material for

comparison, the different varieties of amputations comprised in our 400 cases have been set forth in the following tables :

Amputation at Hip-joint for Disease.

	No. of cases.	Died.	
From 5 to 10	2	1	Hip disease. Death from shock in 2 hours.
From 10 to 15	2	1	Periostitis of femur and disorganisation of hip-joint. Died from exhaustion and vomiting in 21 days.
From 15 to 20	1	1	Similar case to above. Death from shock in 20 minutes.
From 20 to 30	3	2	1. Broncho-pneumonia; lardaceous liver, kidneys, and spleen. 2. Exhaustion; patient in a very weak state at time of operation, to which he had refused consent.
From 30 to 40	1	—	—
From 40 to 50	1	1	Old hip disease. Death from exhaustion in 12 hours.
	10	6	

Mortality 60 per cent.

The number of cases is too small to justify us in drawing any conclusions. From the nature of the cases the large mortality is not surprising, for the operation was in all undertaken more with the object of prolonging or making life more endurable than of actually saving it.

Primary and Secondary Amputations of the Thigh, including Double Amputation.

	No. of cases.	Died.	
Under 2 years	1	1	From pyæmia (Table VI, A, No. 5).
From 5 to 10 years	1	1	From shock.
From 10 to 15 years	3	2	1 from shock and other injuries.
From 15 to 20 years	1	0	—
From 20 to 30 years	3	1	From shock and hæmorrhage from accident.
From 30 to 40 years	8	4	1 from exhaustion, the other leg having been amputated at knee; 1 collapse, loss of blood at time of accident; 1 exhaustion (case of suicide); 1 collapse on operating table.
From 40 to 50 years	8	5	1 on table (both thighs); 1 from shock, hæmorrhage before operation; 1 from pyæmia (?) before operation (Table VI, A, No. 6); 1 from diabetes; 1 from pyæmia following simple fracture into the knee-joint (hæmarthrosis and suppuration) (pyæmia probably preceded operation) (Table VI, A, No. 7).
From 50 to 60 years	8	6	1 from exhaustion; 1 from hæmorrhage at time of accident; 1 visceral disease; 3 from shock, of which 1 died on table.
From 60 to 70 years	1	1	1 from shock.
One man, age unknown, died from shock in a few hours.			
	1	1	
	35	22	

Mortality 62 per cent.

Amputation of Thigh for Disease.

	No. of cases.	Died.	
Under 5 years	6	0	—
From 5 to 10 years	17	0	—
From 10 to 15 years	11	1	From tuberculosis.
From 15 to 20 years	17	6	From phthisis and exhaustion 1; from pyæmia before operation 2; from exhaustion and prolonged suppuration before operation 2; from shock and hæmorrhage in previous sequestrotomy 1.
From 20 to 30 years	32	4	From shock 1; hæmorrhage 2; from pyæmia 1 (Table VI, A, No. 3); from phthisis 1.
From 30 to 40 years	21	1	From exhaustion and erysipelas before operation.
From 40 to 50 years	20	4	From erysipelas 1; from exhaustion 2; from diabetes 1.
From 50 to 60 years	6	3	From exhaustion in case of senile gangrene 1; from tetanus 1; from exhaustion and chronic suppuration 1.
From 60 to 70 years	5	3	From recurrent hæmorrhage and exhaustion 1; from exhaustion and gangrene 1; from exhaustion due to gangrenous cellulitis 1.
Above 70 years	1	1	From gangrene of flaps.
	136	23	

Mortality 16·9 per cent.

These tables call for no special comment, save that they illustrate well the influence of age, which has already been considered.

Amputation through Knee-joint for Disease.

	No. of cases.	Died.
Above 15 years of age and under 20 . .	1 ...	0
„ 20 and under 30	7 ...	1
„ 30 „ 40	5 ...	0
„ 40 „ 50	4 ...	0
„ 60 „ 70	3 ...	3
	20	4

Mortality 20 per cent.

Primary and Secondary Amputations through the Knee-joint, including Multiple Amputations.

Six cases. Four deaths.

In three of these fatal cases the amputation was double.

Amputation through Knee-joint.

Deaths.

1. Lisfranc on other foot at same time; primary amputation; traumatic delirium day after operation. Died of exhaustion after 10 days.
2. Malignant tumour of leg; secondary hæmorrhage.
3. Primary; right thigh amputated above condyles; left through knee. Died of exhaustion after 14 days.
4. Double amputation; primary; right through knee; left leg about middle. Died of exhaustion after 23 days.
5. For old ulcer of leg; recurrent hæmorrhage from stump; sloughing of flap. Died of exhaustion after 40 days.
6. Primary; case of compound fracture; much collapse on admission. Died after 3 days.
7. For senile gangrene, to relieve intense pain; commencing gangrene of flaps. Died of asthenia after 6 days. Pain ceased after operation.
8. Man, æt. 61. Chronic abscess of head of tibia; flaps retracted. Died of pyæmia (Table VI, A, No. 8).

Most of these amputations were performed in the manner advocated by Mr. Stephen Smith. The healing was often slow but the resulting stumps left nothing to be desired.

Amputation of Leg for Disease.

	No. of cases.	Died.	
Under 5 years of age	1	—	—
Above 5 and under 10	6	—	—
„ 10 „ 15	7	—	—
„ 15 „ 20	8	—	—
„ 20 „ 30	8	1	From septicæmia, which existed previous to amputation.
„ 30 „ 40	8	—	—
„ 40 „ 50	9	1	From pyæmia (Table VI, A, No. 1).
„ 50 „ 60	9	2	1 from sloughing and gangrene; 1 from erysipelas.
„ 60 „ 70	2	2	1 from gangrene; 1 from exhaustion.
	58	6	

Mortality 10·3 per cent.

Primary Amputation of Leg, including Double and Multiple Amputations.

Age.	No. of cases.	Deaths.	
Under 5 years	1	0	—
Above 10 and under 20	2	0	—
„ 20 „ 30	6	1	From shock; both forearms also amputated.
„ 30 „ 40	3	1	Exhaustion and gangrene after compound fracture.
„ 40 „ 50	4	0	—
„ 50 „ 60	5	4	2 from shock; 1 from other injuries, and 1 from pyæmia which existed previous to amputation.
„ 60 „ 70	2	2	1 from delirium and exhaustion, and 1 from other injuries.
„ 70.....	1	1	Secondary after compound fracture; sloughing and exhaustion.
Age unknown.....	1	1	Shock.
	25	10	

Mortality 40 per cent.

Syme's Amputation.

Age.	Cases.	Recovered.	Died.
Under 5 years . .	4	4	
Above 5 and under 10	4	3	1
„ 10 „ 85	3	3	
„ 15 „ 20	6	5	1
„ 20 „ 30	14	12	2
„ 30 „ 40	3	3	0
„ 40 „ 50	4	3	1
„ 50 „ 60	3	3	
„ 60 „ 70	3	3	
	44	39	5

Mortality 11·3 per cent.

1. Pyæmia. Died after 20 days. Suppuration of ankle (Table VI, A, No. 2).
2. Carbolic acid poisoning; sloughing of heel flap. No post-mortem examination (Table VI, A, No. 4).
3. Died after 71 days; stump not healed; hæmorrhage from rectum and dysenteric diarrhœa. No post-mortem.
4. Died of phthisis, bedsores, and exhaustion; sloughing of heel flap. No post-mortem.
5. Flap sloughed; urine became albuminous; diarrhœa, ascites, and vomiting. Died in 50 days of lardaceous disease.

Amputation through Shoulder-joint.

Primary for Accident.

	No.	Died.
Æt. 20	1	0

For Disease.

Age.	No. of cases.	Died.	
From 15 to 20 years	1	—	—
„ 20 to 30 „	3	1	Death from shock in 7 hours.
„ 30 to 40 „	3	—	—
„ 40 to 50 „	1	—	—
„ 50 to 60 „	1	1	Death from exhaustion in 5 days.
	9	2	

Mortality 22 per cent.

Amputation of Arm for Accident.

Age.	No. of cases.	Died.	
Under 5 years	1	0	—
From 5 to 10 years	1	0	—
„ 10 to 15 „	—	—	—
„ 15 to 20 „	—	—	—
„ 20 to 30 „	4	0	—
„ 30 to 40 „	3	1	From penetrating wound of chest.
„ 40 to 50 „	3	0	—
„ 50 to 60 „	1	0	—
„ 60 to 70 „	1	1	From shock.
Above 70	1	1	Gangrene and exhaustion.
	15	3	

Mortality 20 per cent.

Amputation of Arm for Disease.

Age.	Cases.	
Above 15 and under 20 . . .	4	All recovered.
„ 20 „ 30 . . .	3	
„ 30 „ 40 . . .	1	
„ 50 „ 60 . . .	2	
	10	

Amputation of Forearm for Disease.

Age.	Cases.	Died.
Between 20 and 30	8 ...	1
„ 30 and 40	7 ...	0
„ 40 and 50	4 ...	0
„ 50 and 60	5 ...	1
„ 60 and 70	3 ...	0
„ 70 and 80	1 ...	0
	28	2

Primary Amputation of Forearm.

Age.	Cases.	Died.
Between 10 and 20	3 ...	0
„ 20 and 30	4 ...	2
„ 30 and 40	1 ...	0
	8	2

1. Both forearms and leg; primary. Died in 10 hours.
2. Deformity from old burn seemed doing well when severe vomiting came on on 117th day and could not be stopped. No post-mortem. ? Died of operation.
3. Stump healed. Died in 49 days. Post-mortem, advanced phthisis.
4. Many other injuries; fracture right femur and left clavicle, scalp wound, and wound of right foot, from which pyæmia developed, of which he died in 7 days. Primary amputation.

We have thought it as well to tabulate these cases, though practically amputation of the forearm *per se* is an operation of infinitesimal risk. In the 'Pathological' series, as will be seen, two deaths occurred. These can only be included in our tables by the necessity for rigid observance of the rule laid down at the commencement of this paper. In one case the patient, who was fifty-five years of age, died from circumstances wholly unconnected either with the operation or the disease that had rendered it necessary. In the other the object of the operation was fully attained, and the patient was relieved of a painful local disease, but died of advanced phthisis.

We may now briefly consider the deaths after operation, and their causes.

Table III furnishes the results of—

(A) Amputation for disease.

(B) Amputation for injury, chiefly primary.

(C) Double or multiple amputations for injury.

In the first section (A) we have 314 cases and 48 deaths, showing a mortality of 15·2 per cent. In the second section (B) 79 cases with 29 deaths, a mortality of 36·6 per cent. In the third section (C) 7 cases and 7 deaths, a mortality of 100 per cent. In some of the cases tabulated under (C) the injuries were not limited to the limbs amputated, and the operations were undertaken with little more hope than that of enabling the sufferers to pass more peacefully out of the world.

TABLE III.

(A) *Amputation for Disease.*

Nature of amputation.	No. of cases.	Deaths.
Thigh	136 ...	23
Thigh at hip-joint . .	10 ...	6
Leg	58 ...	6
Leg at knee-joint . . .	19 ...	4
Syme	44 ...	5
Arm	10 ...	—
Arm at shoulder-joint . .	9 ...	2
Forearm	28 ...	2
	314 ...	48

I. e. 15·2 per cent.

(B) *Primary Amputations.*

Nature of amputation.	No. of cases.	Deaths.
Thigh	32 ...	18
Leg	21 ...	6
Leg at knee . . .	3 ...	1
Arm	15 ...	3
Forearm	8 ...	1
	79 ...	29

I. e. 36·6 per cent.

(C) *Primary Double and Multiple Amputations.*

Nature of amputation.	No. of cases.	Deaths.
Thighs, both . . .	2 ...	2
Thigh and other leg at knee-joint .	1 ...	1
Leg „ „ .	1 ...	1
Legs, both	1 ...	1
Legs at knee-joint and Lisfranc .	1 ...	1
Leg and both forearms . .	1 ...	1
	7 ...	7

I. e. 100 per cent.

Summary.

For disease . . .	314 cases,	of which	48 died.
Primary . . .	79 „	„	29 „
„ double or multiple .	7 „	„	7 „
	400		84

TABLE IV.

From causes unconnected with the operation; death inevitable.

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
1	Leg, secondary	508	37	Limb removed for gangrene following compound fracture. Death in 3 days.
2	Legs, primary	509	50	Railway accident; both legs amputated; never rallied from shock of injury. Death on 3rd day.
3	Thighs, primary	510	13	Both thighs amputated; also compound fracture of arm. Death in a few hours.
4	Leg at knee, primary	513	68	Railway accident; delirium came on one hour after injury; Lisfranc's amputation on other foot. Death in 10 days.
5	Leg, primary	517	28	Railway accident; extreme collapse; both forearms also removed. Death in 10 hours.
6	Thighs, primary	523	44	Both thighs removed. Death on operating table.
7	Leg, secondary	527	57	For suppuration and necrosis in case of compound fracture into ankle-joint 5 weeks previously; pyæmia existing prior to amputation. Death in 7 days.
8	Leg, primary	528	56	Died in 2 days without recovering from original shock.
9	Arm, primary	562	31	Gunshot wound, causing great collapse. Death in 2 days.
10	Thigh, primary	564	38	Other leg removed through knee-joint; railway accident. Death in 14 days.
11	Do.	577	?	Other injuries. Death in 5 hours.
12	Forearm	583	55	Removed for extensive ulceration from burns. Died vomiting on 117th day. No post-mortem. (This case ought probably to be classed as a recovery from amputation.)
13	Thigh, primary	612	13	Also compound fracture of arm. Death in 8 hours.
14	Do.	613	32	Severe loss of blood prior to operation. Death in 48 hours.
15	Do.	619	33	Suicidal injuries. Death in 2 days.
16	Do.	633	35	Railway accident. Death on operating table.
17	Forearm, primary	650	23	Many other injuries, from which pyæmia was developed. P.M.—Multiple pyæmic abscesses. Death in 7 days.
18	Leg, primary	665	?	Both legs crushed. Death in 20 minutes.
19	Thigh, primary	666	3	Collapse from accident. Death in 3 hours.
20	Leg at knee, primary	671	62	Collapse from accident. Death in 3 days.

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
21	Thigh, primary	715	51	Loss of blood prior to amputation. Death in 36 hours.
22	Leg, caries of tarsus	717	31	Died exhausted in 3 days. P.M.—Tubercles in lungs.
23	Thigh, primary	758	55	Other injuries. Death on operating table.
24	Leg, primary	776	53	Other injuries. Death in 2 hours.
25	Thigh, for disease	841	61	Extensive gangrenous cellulitis of leg. Death in 6 hours.
26	Do.	855	50	Extreme exhaustion following suppuration of knee. Death in 8 hours.
27	Thigh, primary	870	58	Railway accident. Death in 8 hours.
28	Do.	876	44	Other injuries. Diabetes. Died comatose in 6 days.
29	Leg, primary	882	68	Fracture of base of skull; also compound fracture of other leg. Death in 2 days.
30	At hip-joint, for disease	886	17	Extreme exhaustion in case of periostitis and necrosis of femur. Patient almost moribund from prolonged suppuration. Hæmorrhage at operation from œdematous tissues. Death in 20 minutes.

Table V.

From other causes coinciding with the operation, the other causes having a main share in producing death.

Class A.—*Death due mainly to previous visceral or local disease.*

1	Leg, for senile gangrene	507	70	Gangrene attacked the flaps, and death occurred on 5th day. P.M.—Atheromatous vessels.
2	Thigh, for diseased knee	533	16	Tubercular child; very feeble from prolonged suppuration. Death on 4th day.
3	Do.	534	13	Suppuration of knee. Death in 11 days. P.M.—Tubercles in lungs.
4	Thigh, for failure of excision of knee	545	15	Died in 26 days, worn out by pain and suppuration following the excision.
5	At hip-joint	568	25	Operation previously declined; exhausted at time of amputation. Sank in 6 days.
6	Thigh, for pulpy disease of knee	574	22	Died of phthisis in 82 days.
7	Do.	575	46	Diabetes. Died in 12 days.
8	Forearm, for diseased wrist	604	28	Died in 49 days. P.M.—Advanced phthisis.

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
9	Syme, for disease of ankle	644	34	Wound not quite healed when man died on 71st day from hæmorrhage from rectum and dysenteric diarrhœa. No P.M.
10	Syme, for disease of tarsus	648	19	Phthisis. Heel flap sloughed. Death in 9 days from exhaustion and bed-sores. No P.M.
11	Thigh, for sarcoma of tibia	651	36	Myeloid sarcoma of tibia gouged out, followed by erysipelas and suppuration of the knee-joint. Death on operating table.
12	Thigh, for suppuration of knee	693	16	Phthisical patient. Acute periostitis of tibia, followed by destruction of knee-joint. Death in a few days. No P.M.
13	Do.	699	18	Suppurative arthritis of knee. Pyæmia before amputation. Death in 10 days.
14	Leg, for sloughing	702	54	Excision of os calcis was followed by sloughing of soft tissues of opposite heel. Diseased arteries. Death in 21 days.
15	At hip-joint	726	13	Periostitis of femur and suppuration of joint. Death in 21 days from exhaustion. P.M.—No morbid change found in internal organs.
16	Thigh, for diseased knee	732	15	Pyæmia and disorganisation of knee followed aspiration of joint for synovitis. Death in 41 days. P.M.—General tuberculosis.
17	Leg and knee, for gangrene	773	66	Operation to relieve pain in case of senile gangrene. Death on 6th day. P.M.—Embolism of mid-cerebral artery; atheroma.
18	Thigh, for gangrene	799	67	Death in 10 days. P.M.—Arterial degeneration and chronic interstitial nephritis.
19	At hip-joint	802	48	Prolonged suppuration of hip; bed-sores. Death in 12 hours.
20	Syme, for disease of tarsus	829	9	Death in 50 days from lardaceous degeneration of viscera.
21	At hip-joint, for disease of joint	830	23	Death in 27 days from broncho-pneumonia and lardaceous disease.
22	Leg, for senile gangrene	857	64	Amputated for severe pain. Gangrene attacked the flaps, and extended to the groin. Death in 11 days.
23	Thigh, for senile gangrene	899	54	Senile gangrene; thrombus in femoral artery at site of amputation. Death in 7 days. P.M.—Thrombosis of left pulmonary artery and external iliac artery.

CLASS B.—*Death due mainly to the consequences of previous injury.*

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
1	Leg, secondary	611	28	Railway accident. Right leg removed through the knee, and the left below the knee; sloughing of flaps and exhaustion. Death in 21 days. No P.M.
2	Thigh, secondary	632	55	For gangrene following primary excision of ankle-joint for compound dislocation. Drayman, in a low state. Death in 24 hours.
3	Thigh, for ruptured popliteal artery	641	58	Severe hæmorrhage at operation; great extravasation into soft tissues of limb. Death in 3 days.
4	Thigh, secondary	667	57	For sloughing after compound fracture. Died suddenly on 3rd day. P.M.—Fatty degeneration of cardiac muscles and granular kidneys.
5	Do.	688	41	After compound fracture; a piece of necrosed bone ulcerated into the popliteal artery; severe hæmorrhage and collapse. Death in 2 hours.
6	Leg, secondary	757	70	Compound fracture; flaps sloughed. Phlebitis and delirium. Death in 9 days. No P.M.
7	Thigh, secondary	787	50	Secondary to compound fracture; septicæmia. Death in 25 days. P.M.—No pyæmic deposits.
8	Thigh, primary	823	28	Severe hæmorrhage before admission. Death in 10 hours. (Possibly ought to be in Table I.)
9	Thigh, ruptured posterior tibial artery	854	66	Enormous extravasation of blood into leg. Death in 30 hours.
10	Arm, secondary	874	68	Commencing gangrene after compound fracture opening the elbow-joint. Death in 5 days.

TABLE VI.

Deaths from amputation, i. e. due mainly to the consequences of the operation itself.

CLASS A.—*From Pyæmia.*

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
1	Leg	526	44	R. A. had caused dislocation of the ankle and rendered the limb useless. Rigors on 5th day after amputation, and death from pyæmia followed on the 22nd day.
2	Syme, for disease of foot	536	41	Death from pyæmia in 20 days. Pyæmia began on the 17th day.
3	Thigh, for disease of knee	550	23	Death from pyæmia in 9 days. No P.M. examination.
4	Syme, for disease of tarsus	635	25	A delicate man with cardiac disease. Carboluria followed amputation. Septicæmia and sloughing of heel flap. Death in 10 days.
5	Thigh, secondary	716	1½	For gangrene following a simple fracture of thigh. Death from pyæmia in 17 days. There was also a wound of the other foot. (Pyæmia probably preceded the amputation.)
6	Do.	723	43	For compound fracture. Death from pyæmia in 12 days. (Pyæmia almost certainly preceded operation.)
7	Do.	833	49	Secondary for suppuration of knee following hæmarthrosis in case of fracture of femur into joint. Septicæmia. Death in 9 days. (Pyæmia probably preceded operation.)
8	Leg at knee, for disease of tibia	834	61	For chronic osteo-myelitis of tibia. Rigors and pyæmia, and death in 10 days. P.M.—Abscess in spleen and thrombosis of femoral vein.

CLASS B.—*Sloughing (in Mr. Holmes's papers this class included also Phagedæna).*

1	Thigh, for ulcer of leg	652	68	Recurrent hæmorrhage; sloughing of flaps; bedsores; exhaustion. Death in 40 days.
2	Thigh, for diseased knee	751	50	Sloughing and gangrene of flaps; osteo-myelitis; tetanus. Death in 19 days.

Class C.—*From Erysipelas.*

No.	Nature of amputation.	Amp. book No.	Age.	Remarks.
1	Thigh, for ankylosed knee	504	46	Died in 62 days from erysipelas, exhaustion, and bedsores.
2	Leg	682	55	Amputated for old injury to foot. Erysipelas attacked the wound within 24 hours of the operation. Sloughing of flaps and bedsores. Death in 12 days.

Class D.—*From Secondary Hæmorrhage.*

1	Arm, secondary	516	71	For gangrene following fracture of surgical neck of humerus. Secondary hæmorrhage on 6th day. Vessels very atheromatous. Death on 7th day.
2	Leg and knee for malignant tumour of leg	555	22	Profuse secondary hæmorrhage. Death in 17 days.

Class E.—*Shock at Operation; hæmorrhage.*

1	Thigh	521	27	Thigh removed for myeloid sarcoma at lower end of femur. Death in 18 hours from shock.
2	Do.	640	43	Ununited fracture of femur. Death from shock of operation on 3rd day. P.M. — Vessels atheromatous; heart fatty; emphysema of lungs; fibroid and fatty changes in liver.
3	Arm at shoulder	741	25	For sarcoma of humerus. Profuse hæmorrhage and shock. Death in 7 hours.
4	Thigh	760	40	Amputated for secondary hæmorrhage after ligature of femoral artery for wound. Shock. Death in 2 days.
5	Arm at shoulder	761	58	For sarcoma of humerus. Severe hæmorrhage at operation. Death in 5 days.
6	Thigh	821	17	For periostitis and necrosis of tibia. An attempt was made to remove the shaft of the tibia; this was followed by profuse hæmorrhage, and the limb was at once removed. Shock. Death in 3 days.
7	Do.	826	29	Sarcoma of muscles of lower third of thigh. Profuse hæmorrhage; large muscular thigh. Shock. Death in 40 hours.

Still following Mr. Holmes' method of arranging the cases, we have drawn up a complete list of all the deaths. These Tables IV, V, and VI speak for themselves. The interest chiefly centres in the last two. It will be understood that our main object has been to afford a ready comparison with the previously published statistics drawn from the same source. Exception may be taken to the headings under which particular cases have been entered, but it has been our aim to secure uniformity even though some sacrifice should be involved in other respects. No branch of medical literature has been more fiercely criticised than the statistical. Undoubtedly the material is somewhat plastic, but much of the distrust excited by any statistical paper is due to two causes: one, the want of uniformity in statistics furnished from different quarters; the other, that advertisement so often unfortunately adopts the statistical guise. At the least we will endeavour to avoid the former of these two drawbacks. It is often said that "anything may be proved by statistics," and the statement is intended to imply distrust. It may be so; it is also difficult to prove anything in any other way.

Table IV comprises the deaths occurring from causes unconnected with the operation, and in which death was really inevitable. Thirty cases are included, twenty-six of these being primary or secondary amputations for injury. One of the deaths occurred in a patient whose forearm was amputated for extensive ulceration the result of a burn. This patient was attacked with severe vomiting and died on the 117th day after operation; no post-mortem examination was allowed. As regards the operation she had really recovered, but for reasons assigned already the case is entered here as a death after amputation.

Table V includes the deaths occurring from other causes coinciding with the operation, the other causes having a main share in producing death. These cases are divided into two classes.

Class A. *Death due mainly to previous visceral or local*

disease.—In this class we have twenty-three cases. In seven of these death was due mainly to phthisis. Four were cases of senile gangrene. Two died of pyæmia which existed markedly before operation. One man aged thirty-four, on whom a Syme's amputation had been performed, died on the seventy-first day from hæmorrhage from the rectum and dysenteric diarrhœa. No post-mortem examination was allowed. The wound had not quite healed.

Class B. *Cases due mainly to the consequences of previous injury.*—Ten cases, all primary or secondary. In one case septicæmia developed after compound fracture, and the limb was amputated. Death in twenty-five days. Post mortem no evidence of pyæmia was found. Some of these cases might perhaps more properly be transferred to Table IV.

Table VI includes the cases in which death may really be ascribed to the effects of the operation, or, if the term be preferred, from surgical calamities. Altogether twenty-one deaths are tabulated. Two patients died from sloughing (one of these from tetanus): in Mr. Holmes' 500 cases seven are recorded from similar causes. Two died from secondary hæmorrhage: in the previous tables four are entered. Seven died from shock and loss of blood at the amputation. Two died from erysipelas: in the previous tables five.

Finally, from pyæmia our tables (Table VI, A) show eight cases in 400 amputations; though, as will be mentioned directly, the number might more properly be reduced to four. For the purposes of this table we draw no distinction between septicæmia and pyæmia.

The previous tables show seventy-five deaths from pyæmia in 500 amputations. The contrast is in itself so striking that it needs but little comment. Cases in which pyæmia unquestionably had set in before amputation are not included. In two of our eight cases (5 and 6) pyæmia almost certainly preceded operation, and in one other (7) it probably did so. Yet it has been thought better to tabulate these cases under the pyæmia heading. In the

case of one patient (4), on whom a Syme's amputation had been performed, the heel flap sloughed. This man, who was the subject of cardiac disease, suffered also from carboluria. German surgeons would have set down the death probably not to septicæmia, but to carbolic poisoning.

In Cases 1, 2, 3, and 8 pyæmia seems undoubtedly to have resulted from the amputation. It is noteworthy that two cases occurred in 1876, one in 1877, one in 1880, two in 1883, and finally two in 1886, in patients who were submitted to amputation within three days of each other—a significant fact. At the least this occasional occurrence of pyæmia shows that the battle was not being waged against any imaginary foe. The conditions, whatever they be, tending to the production of pyæmia were there, and were only kept at bay by adequate precautionary measures. To our minds there is absolutely no question that the whole of the improvement as regards the pyæmia is due to the careful carrying out of the antiseptic system. It is a preventable disease, and, as the figures show, has almost entirely been prevented.

Amputations have been taken as a convenient example only of major operations. Statistics of other operations would show the same results. All the details necessary for the thoroughly efficient carrying out of the antiseptic system of surgery can be best practised in hospitals, and it is not, perhaps, too much to assume that, considering the extremely minute risk shown to be run in a well-managed London general hospital, the old spectre of "hospitalism" may be said to be laid.

At the outset of this paper it was pointed out that from 1874 to 1879 inclusive the methods were constantly undergoing slight changes, and that in our tables, unfortunately for purposes of comparison, we are not able to define strictly what may be termed an antiseptic period. Yet it is very significant that the percentage of mortality from 1874 to 1879 inclusive was 26·3 for all cases, while from 1880 to 1888 it fell to 18·8.

If we exclude the cases of death already tabulated as

not really due to amputation, we are left with twenty-one deaths in 341 cases, or a mortality of about 6 per cent. We believe that some such figure represents the real risk of the occurrence of a surgical calamity in an average amputation.

To sum up, then, amputation is practically an operation almost devoid of risk in a selected case, and a person under fifty years of age. After that period of life the danger is greatly increased, though not to the extent shown in the tables, for the gravity of diseases in the old requiring the performance of an amputation chiefly contributes to the increased mortality. Still the risk is undeniably very much greater, even though the dangers arising from degenerated blood-vessels are sensibly diminished by the modern forms of ligatures.

In conclusion we have to thank the members of the surgical staff of the hospital for the ready permission accorded to make use of their cases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ii, p. 136.)

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